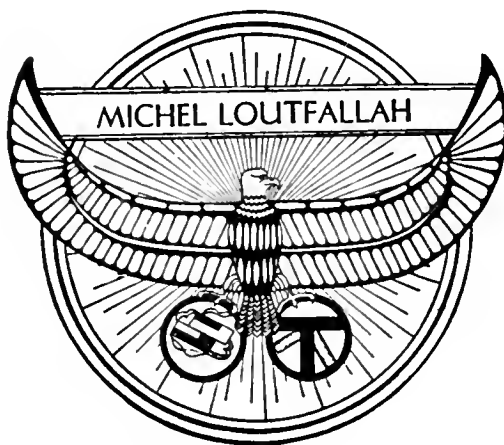




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ATLAS OF EXTERNAL DISEASES OF THE EYE

BY

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**WITH 84 COLORED ILLUSTRATIONS FROM WAX
MODELS PRINTED ON 54 PLATES WITH
EXPLANATORY TEXT**

THE MODELS FROM WHICH THE ILLUSTRATIONS WERE MADE ARE IN THE
PATHOPLASTIC INSTITUTE IN BERLIN; F. KOLBOW, Art Director



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EDITOR'S PREFACE

AT THE request of the publisher I have undertaken the difficult task of trying to reproduce in English *Greeff's* beautiful Atlas, and at the same time to render the work as a whole of more utility to the general practitioner. In many places in the original text the reader receives the impression that the author has been cut short in what he wished to say, perhaps by the limitations of space, perhaps by the feeling that he was writing for specialists who were quite well acquainted with the subject. This is particularly true of treatment, which is often dismissed with a few words. I have therefore added much of detail, and have tried to incorporate the latest advances in this branch of ophthalmology. In order that no confusion may arise between the work of the distinguished author and that of the humbler editor, I have enclosed in brackets [] everything that is not to be found in the original text.

M. L. FOSTER.

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Fig. 1.
Erysipelas Faciei Oedema palpebrarum

Facial Erysipelas. Oedema of the Eyelids.

Plate I, FIG. 1

Facial **erysipelas** usually follows some slight injury of the skin that gives entrance to the streptococcus erysipelatis. It is characterized by a high fever, sometimes accompanied by chills, and the development of a sharply defined, glistening red swelling of the skin which spreads more or less rapidly until it may have extended over the entire face, and presents on its surface a larger or smaller number of vesicles. [These *streptococci*, which are nearly related to streptococci pyogenes, are to be found in the lymph spaces of the skin during the early stages of the inflammation, but as a rule have disappeared by the time the disease has reached its height. Few or none are to be found in the contents of the vesicles, and they are rarely present in the blood, except when suppuration has taken place. Sometimes the local lesion and the general symptoms appear together, in other cases there is an **incubation period** of from one to three days, characterized by headache, malaise, and anorexia. The attack usually begins with chilliness or a chill, followed by fever, the temperature ranging from 102° to 105° F. The skin affected has a well defined zig-zag border, is hot to the touch, tender, firm and smooth, and is the seat of a sensation of tension, heat, burning, throbbing pain, tingling, or a mild pruritus. The redness fades under pressure, leaving a yellow stain. The vesicles sometimes coalesce into large bullae. The maximum extent and severity of an attack is reached in about a week. The cervical lymphatic glands are moderately swollen, but the swelling is hidden by the oedema of the neck. A moderate *leucocytosis* is usually present, 7,000 to 8,000 in mild cases, 12,000 to 20,000 in severe, and even 40,000 to 60,000 when suppuration has taken place. Generally it varies with the fever, but it sometimes is absent even when the fever is high. An almost constant feature is the *gastro-intestinal disturbance*; the tongue is coated, the appetite lost, frequently there is vomiting, and

the bowels may be constipated or loose. The spleen is moderately swollen. The patient usually suffers from headache and prostration. The constitutional symptoms are due largely to toxins, the visceral lesions that occasionally form complications, to secondary metastatic infections.]

The **eyelids** become greatly swollen, often the eyes cannot be opened by the second day. The skin of the lids becomes very red and bullae are particularly apt to form at their margins (Fig. 1, right eye). The great abundance of inflammatory oedema is due to the fact that the skin of the lids is attached to the subcutaneous tissue more loosely than it is elsewhere, and that no subcutaneous fat is present, so that space is afforded for the accumulation of fluid.

After a few days the fever begins to abate, the bullae rupture, the redness and oedema of the skin diminish, and desquamation and pigmentation appear over the affected area.

Diagnosis

The diagnosis is usually easy. The onset of fever associated with a circumscribed area of elevated, red and shining skin, with its advancing, irregular margin, is so characteristic that a mistake can seldom be made.

Prognosis

The prognosis is usually good, yet the disease must be regarded as serious. A general infection, evidenced by high fever and very great prostration, may prove fatal. In some cases there is an inflammation of the subcutaneous connective tissue that may result in abscess or gangrene of the lid. [Whenever an abscess can be detected in the lid it should be opened freely, for otherwise it may extend deeply into the orbit and cause death by meningitis.]

Sudden relapses are apt to occur during convalescence, and the disease predisposes its victims to new attacks for a long time. [The fact that it confers no immunity upon the organism has an important bearing upon the probable benefit to be obtained from serum treatment.]

Treatment

[The treatment of erysipelas may be divided into local and general. No **local treatment** has much power to check or modify the course of the disease, but something may be done to contribute to the comfort of the patient. Cold water has been used for this pur-

pose from the days of Hippocrates, but hot fomentations are more grateful in many cases. A cold or hot compress may be made by wringing cloths out of solutions of bicarbonate or hyposulphite of sodium, of boric acid, or of salicylic acid, or a lead and opium wash may be used. The skin may be anointed with oil or vaseline to soften it and relieve its tension, or an ointment containing boric acid, resorcin, or ichthyol (2% to 5%) may be used for the same purpose. Or the surface may be dusted with a simple powder, such as starch, iodoform, or sulphur, and then covered with a layer of soft cotton. Iodine may be painted outside the elevated border of the inflamed skin to excite an active hyperæmia and so establish a barrier of leucocytes against the further advance of the disease, but this measure does not always prove effective. The benefit to be derived from *Bier's* congestion hyperæmia is still questionable. When the tension of the lids becomes menacing and the patient is in great pain it is sometimes necessary to scarify the skin; in such cases the incisions should always be made in the long diameter of the lid.

The **general treatment** is mainly hygienic and symptomatic. The patient should be kept in bed in a well ventilated room, the temperature of which should be maintained uniformly between 65° and 70° F., and isolated, as in other contagious diseases. All dressings that have come in contact with his skin should be burned, and the sick room, with all its accessories, should be thoroughly disinfected before it is used again by other persons. The body temperature may be controlled by baths or wet packs if it becomes excessive, but usually it responds sufficiently to iced or acid drinks, or to some mild febrifuge, while strength should be maintained by good nursing and a nutritious, light diet.

The tincture of the chloride of iron, in doses of from twenty to forty drops well diluted with water every two or three hours, has enjoyed a high reputation in the United States; sometimes it seems to shorten the attack, but in other cases it has no appreciable effect, and it does not reduce the mortality. Its real value is questionable, because attacks of this disease vary so much in their virulence and duration.

Antistreptococcic serum and streptococcus vaccine are still on trial, and it remains to be learned whether either will lessen the rate of mortality, or the duration of the attacks. Too much must not be expected from this form of treatment as the disease confers no immunity.

Intravenous injections of collargol have been recommended in

grave cases, and inunctions of colloidal silver ointment are said to have been beneficial.]

OEDEMA OF THE EYELIDS

This is not a disease *per se*, but a secondary phenomenon observed in many affections. Effusions of fluid find plenty of space in which to accumulate beneath the loosely attached skin of the lid, and they are quite apt to be large. [The condition can always be recognized from the fact that the skin pits on pressure with the finger.]

A common example of the **traumatic form** is the ordinary “*black eye*” (see Fig. 2), but oedemas so great as to cause the lids to look like distended bags and to completely close the eye may be excited by such slight injuries as bee stings, flea bites, and local poisonings. Oedema accompanies not only such *acute affections of the lids* as abscess, hordeolum, and erysipelas, but also *inflammations in the neighborhood*, like dacryocystitis, disease of the accessory sinuses, periostitis of the wall of the orbit, orbital cellulitis, blennorrhea, panophthalmitis, and other severe acute diseases of the eye.

It is well known that ***anasarca*** due to renal or cardiac disease, or to a hydraemic condition of the blood, is very apt to show itself in the eyelids at the same time that it causes a swelling about the ankles.

A doughy oedema of the lids occurs in ***trichinosis***, because the trichinae are apt to settle in the orbicularis.



Fig. 2.
Haemorrhagia subdermalis et subconjunctivalis.

Subdermal and Subconjunctival Hemorrhage.

Plate II, FIG. 2

A **subdermal hemorrhage**, also known as an ecchymosis, a sugillation and a suffusion of the lids, [the ordinary "black eye,"] is an extravasation of blood beneath the skin of the eyelids, which is attached so loosely to the subjacent tissues that blood accumulates and spreads beneath it very readily. The limits of the hemorrhage usually are sharply defined at the margin of the orbit, where dense connective tissue joins the skin firmly to the bone, but it is quite apt to spread over the dorsum of the nose and to appear in the lids of the other eye. The skin over the nose is thick enough to conceal the blood beneath it, so that no connection can be seen between the two black eyes, and when this happens the observer must not be misled into thinking that both eyes have been injured.

The most common **cause** of a subdermal hemorrhage in the lids is a blow from a blunt object, such as a fist, or a club, but its size may not be proportional to the injury inflicted, as very slight blows may suffice to produce large extravasations in persons who suffer from the hemorrhagic diathesis. It is seen after operations in which the free escape of the blood is impeded, as after enucleations of the eye, but seldom after incised wounds in which the blood flows away freely. It is sometimes met with after accidents in which the body has been strongly compressed, and occasionally after violent exertions, coughing, or sneezing, as the lack of supporting pressure about the subcutaneous vessels of the lids renders them more liable to rupture than those in most of the other parts of the body. It is also to be found in cases of fracture of the orbit, or of the base of the skull, when the blood travels forward along the floor of the orbit from the seat of fracture to appear, after a while, beneath the conjunctiva (see below) and the skin of the lower lid, especially near the inner canthus. In such cases the ecchymosis may be of great diagnostic importance.

Diagnosis

The diagnosis of a subdermal hemorrhage is made at a glance from the discoloration of the skin; the only thing that needs to be sought for is its cause.

Prognosis

As a rule the prognosis is good. The red color changes after a short time into a reddish blue, then into a blue black, then into a greenish hue, and the blood is absorbed in the course of a few weeks. Sometimes suppuration takes place and an abscess of the lid develops, when pyogenic organisms have been admitted through a solution of continuity in the skin.

Treatment

[Because of the disfigurement it occasions, every means should be taken to prevent the accumulation of blood beneath the skin of the lid. After an operation for chalazion a black eye can often be avoided by systematically *bathing the eye with as hot water* as can be borne for a few hours. Many prefer *cold*, preferably in the form of ice compresses. Strips of cloth, or pledgets of gauze, are allowed to freeze to the surface of a piece of ice, are then torn off, one at a time, laid on the eye and changed every few seconds. This may be done almost continuously for a few hours, or they may be applied for from ten minutes to half an hour at intervals of an hour. After the extravasation of the blood has ceased, hot compresses are indicated to promote its absorption. Gentle massage is sometimes useful for the same purpose, and so is a pressure bandage. Sedative lotions may be used sometimes with benefit; the best of these are liquor ammonii acetatis, either alone or diluted to half strength, an aqueous solution of ichthyol, 3% to 10%, and a 15% solution of ammonium hydrochlorate in $\frac{1}{3}$ alcohol and $\frac{2}{3}$ water. Calamine ointment, toned down with powder to match the color of the surrounding skin, may be applied over the surface to improve the cosmetic appearance.]

A **hemorrhage beneath the conjunctiva bulbi**, which is attached very loosely to the eyeball, is known as a haemorrhagia subconjunctivalis, a hyphaema conjunctivae, an ecchymoma subconjunctivale, an ecchymosis conjunctivae, and a hyposphagma. It is seen very often in children suffering from **whooping cough**, in whom it is caused by the hard coughing and choking. In older persons it may also be caused by violent exertion, sneezing, vomiting, or straining. It is a common accompaniment of acute inflammation of the

conjunctiva, is produced by the same causes as subdermal hemorrhages in the lids, such as blows, severe compressions of the thorax and abdomen, fractures of the orbit and of the base of the skull, and occurs **spontaneously** in elderly people, when it is indicative of very fragile blood vessels and may be a symptom of arteriosclerosis, Bright's disease, scurvy, or cholera. Hence subconjunctival hemorrhages may have a very serious significance.

Diagnosis

Aside from the determination of its cause the diagnosis is easy. The uniform superficial redness once seen can hardly be mistaken for an inflammation of the conjunctiva, in which the vessels are visible.

Prognosis

The prognosis is good, so far as the hemorrhage itself is concerned. Its sudden appearance is apt to frighten the patient and his friends, but they can be reassured by the statement that the eye is never endangered thereby. One thing must be borne in mind, however, that the spontaneous appearance of a subconjunctival hemorrhage is sometimes a forerunner of cerebral apoplexy; the constitutional cause should therefore always be ascertained and treated.

Treatment

Local treatment is scarcely needed. [Still it is well to apply a lead and opium wash, or cold compresses. Absorption may, perhaps, be hastened in some cases by bathing the eye systematically with hot water.]

Measles. Exanthematic Blepharconjunctivitis.

Plate III, FIG. 3

The catarrhal symptoms that commonly accompany the onset of various acute infectious general diseases are particularly characteristic of **measles**, in which a more or less severe conjunctivitis and blepharitis, with redness, photophobia and secretion, are added to the catarrhal inflammation of the nose and upper air passages, and may precede by several days the outbreak of the eruption. The secretion may consist of an abundant flow of tears, or it may be mucous, or purulent, and dry into crusts on the margins of the lids (see Fig. 3). Croupous membranes are rarely formed.

Prognosis

The acute conjunctival catarrh, which is never absent in measles, usually disappears of itself in the course of two or three weeks, but, if neglected, it may lead to a chronic redness and sensitiveness of the conjunctiva and of the edges of the lids that may persist for years, or for life; hence its treatment should not be slighted. Serious complications that occasionally occur are a blennorrhoeal or diphtheritic conjunctivitis, infiltration of the cornea, and secondary iritis.

Treatment

As with measles in general, cleanliness plays an important part in the treatment of the eyes. Ordinarily it is sufficient to wash the eyelids very carefully with lukewarm boric acid solution as often as may be needed to soften and remove the dried secretion. When the lachrymation is profuse a drop of a weakly astringent collyrium may be placed daily in each eye. Solutions of tannic acid, 1%, of resorcin, 1%, or of zinc sulphate, $\frac{1}{4}\%$, may be used for this purpose. [When the secretion is purulent, or a croupous membrane has formed, an examination should be made at once to determine the active agent, which is the streptococcus in most cases of croupous conjunctivitis that occur in measles. Other agents that are sometimes met with are



Fig. 3.
Morbilli. Blepharo-Conjunctivitis exanthematica.

the pneumococcus, the diphtheria bacillus, the gonococcus, the *Koch-Weeks'* bacillus, the staphylococcus, and the bacillus coli. Not only is the streptococcus the most common agent, but it, either alone or in combination with the diphtheria bacillus, is responsible for the most virulent forms of croupous conjunctivitis in which the conjunctiva becomes necrotic and the cornea is destroyed. When it is present active measures are imperatively demanded. A large dose of antistreptococcal serum, or of vaccine, should be administered with the least possible loss of time. The boric acid solution used for cleansing the eye may be replaced by a solution of the serum, and dry serum may be dusted on the surface of the conjunctiva. A similar procedure may be followed when the agent is any other that is amenable to serum or vaccine therapy. When the agent is not amenable to this form of treatment we must be content with cleansing the eye, removing the cast-off pieces of membrane, and protecting the other eye from infection until the formation of membrane has ceased, the conjunctival secretion has become greater, and the lids have become movable. Then a $\frac{1}{10}$ to 1% solution of silver nitrate may be applied to the conjunctiva and neutralized immediately by a solution of salt. In such cases as these the eye must be watched closely for the appearance of a pericorneal injection, or an infiltration of the cornea, either of which demands the immediate use of a $\frac{1}{2}$ to 1% solution of atropine two or three times a day.]

The sick room should be darkened moderately and the child should not be exposed to the bright daylight until after the signs of irritation have disappeared from the eyes.

Malignant Pustule. Anthrax. Gangrene of the Lids.

Plate IV, FIG. 4; Plate V, FIG. 5

The **malignant pustule**, or the pustule of anthrax, appears in man not infrequently on the lids, where the bacilli from diseased animals have been inoculated by wiping, rubbing, or scratching with the fingers. It is met with chiefly among persons who have to deal with animals or animal products, such as cattle dealers, butchers, tanners, furriers, and workers in other occupations in which the direct handling of animal products is necessary.

The disease frequently starts at the margin of the lid as a [little, itching, burning, red spot which passes through the stages of papule and vesicle within a few hours to become a] pustule filled with yellow, turbid, or bloody contents. This bursts within about 30 hours, leaving a dark red, indurated, comparatively painless nodule with a craterlike excavation which becomes covered by a scab. The adjacent skin is at first livid and red, but soon becomes gray, a sign of commencing necrosis. [Vesicles similar to the first appear upon it and pass through the same stages. In this manner the disease spreads and a rapid destruction of tissue takes place.] The lids are very oedematous, the skin is hard and infiltrated, the preauricular and submaxillary lymphatic glands soon become swollen. [The fever may be slight at first, but it rises rapidly, often to a high degree and is attended by great prostration. The severity of the general symptoms depends on the extent to which the bacilli have invaded the organism and on their virulence.

Malignant anthrax oedema of the lids differs from malignant pustule mainly in the absence of the preliminary vesicle, of the hard nodule, and of the early circumscribed gangrene. The color of the skin is at first pale, and its consistence doughy; later the lid becomes cyanotic, bullae appear and patches of necrosis are formed. This oedema may appear as a sequel to, instead of a precursor of the general disease.]

Vesicles similar to those of anthrax sometimes develop in



Fig. 4.
Pustula maligna — Anthrax.



Fig. 5.
Gangraena palpebrarum-- Anthrax.

phlegmonous inflammations of the lid, carbuncles and glanders, and oedematous anthrax may resemble erysipelas somewhat, so the diagnosis can be established in doubtful cases only by examination for anthrax bacilli, which stain readily and may be discovered at once on a slide. These bacilli are motionless rods with sharp angular corners, usually arranged in chains. [each with a clear open space in its center that corresponds to a spore which is very resistant to heat and desiccation. The occupation of the patient is a valuable guide to the diagnosis.]

Prognosis

The prognosis is bad. [The mortality varies but seems to be at least 25%, and] when recovery takes place the patient usually loses a large portion of his lids from necrosis. [This may, perhaps, be reduced by the use of serum, as *Sclavo* states that the mortality in 164 cases of human anthrax treated in this manner was reduced to 6.09%.

Prophylaxis

Prophylaxis is difficult. The bodies of infected animals should be burned. Rubber gloves would be some protection to workmen, when they can be worn, but they interfere with the sense of touch which is needed in many cases. Disinfection has not yet proved practical. The best that can be done as yet is to try to teach persons who deal with animals and animal products how best to avoid infection while handling them.]

Treatment

Local surgical treatment, [such as incision, curetting with a sharp spoon, or the actual cautery followed by injections of a 5% solution of carbolic acid into the surrounding tissue, which is recommended by high authority for anthrax in general,] is not advisable when the lesion is in the eyelid. We have learned that the less the site of the lesion is disturbed the less will be the danger that the bacteria will enter the circulation. Hence the only local treatment in malignant pustule of the lids should be the application of soothing ointments, or aseptic compresses, while the scabs and necrotic tissue are allowed to separate spontaneously.

[Anthrax serum should be injected intravenously and in the vicinity of the infection. *Sclavo* injected 10 c.c. in each of three or four places subcutaneously, and in grave cases 10 c.c. intravenously;

as much as 150 c.c. may be used at one time. When large doses are given the ether that is employed as a preservative should be caused to evaporate before the serum is used. The treatment may be repeated in 24 hours or sooner if necessary; no deleterious effects have been observed, and it seems to furnish the best hope of success of any method yet known.

When the serum cannot be obtained injections of a 5% solution of carbolic acid may be made frequently about the base of the pustule for the purpose of retarding the multiplication of the bacilli.

The strength of the patient is to be sustained by good nursing, food that is nutritious and easily digested, and the administration of such stimulants and drugs as seem to be indicated.]

Fig. 4 portrays a case of malignant pustule of the lids, for the reproduction of which we are indebted to Prof. *Bockenheimer*. The case was one of external anthrax that developed from a slight scratch on the cheek of a workman in a tannery. A red papule came first, then several vesicles filled with a yellow fluid holding anthrax bacilli, and simultaneously a widespread infiltration like that of carbuncle, a great oedema of the lids, and an erysipelatous redness of the entire cheek. The vesicle at the site of infection soon ruptured and a scab was formed which was surrounded by a gray areola of skin that gradually became necrotic. The process then extended to the lids, which became swollen so enormously that the eye could no longer be opened even by force. The serious systemic involvement was shown at the same time by fever, chills, and delirium. Pustule after pustule developed and were followed, after rupture, by a corresponding gangrene of the skin. The entire side of the face was kept covered by an ointment.

Fig. 5 shows the appearance of the same patient some weeks after the infection. The extensive gangrene, which may be recognized from its black color and leathery condition, is demarked by a zone of pus and dirty granulation tissue from the neighboring skin, which is slightly reddened and inflamed, although the slough is still attached to the subjacent tissue. The forcible removal of this slough by a knife or otherwise would have resulted in a rekindling of the infection. The surface was kept covered with ointment and with compresses wet with hydrogen dioxide and boric acid solution, beneath which separation gradually took place. The entire upper lid sloughed away, leaving a large defect which was filled, after the inflammation had subsided, by a pedicled flap taken from the neighboring skin.

This patient recovered in spite of the bad prognosis in anthrax of the face and in spite of the severity of the local process.

GANGRENE OF THE LIDS

The skin of the lid readily becomes the seat of malignant inflammation and nutritive disturbance because of its delicacy, its thin corium, and its loose subcutaneous tissue, which is provided with large lymph spaces and a rich vascular supply. When the constituent parts of tissue break down with decomposition and putrefaction we say that gangrene is present. **Gangrene of the lid** is therefore a circumscribed fetid necrosis of its tissue surrounded by an areola of inflammatory reaction. [It may be dry or moist; in the moist variety the skin of the lid ulcerates, in the dry it is changed into a black crust.]

According to *Roemer*, who has made a study of this subject, gangrene of the lid may develop endogenously, that is by metastasis, or ectogenously from some local affection.

I. The **endogenous**, or metastatic, form of gangrene occurs infrequently in grave general diseases, particularly in typhoid fever, measles and scarlet fever. As long ago as 1794 *Himly* described a bad case of typhoid fever in which the eyelids became blue and gangrenous within a few hours. *Fieuzal* reported three cases of gangrene of the lid in measles, and similar cases have been recorded by *Knies* and *Randall*. Partial gangrene of the lid has been described by *St. Martin* and *Jackson*, while numerous abscesses of the lid have been observed in influenza. It is caused by infective emboli in pyaemia and sepsis, and has been attributed to diabetes and alcoholism, but all of these cases are rare.

II. In the **ectogenous** form the necrosis may start from a focus of inflammation in the vicinity of the eye, or may develop primarily in the lid itself. Perhaps, it is seen most often as a complication of facial erysipelas. The cause may either be a minute infected wound, or a more extensive injury. *Schmidt-Rimpler* tells of a case in which a swelling of the lids followed a blow from a bough of a tree upon the malar bone; five days later the skin of the lid had been transformed into an ulcer full of necrotic shreds of tissue.

Anthrax is prominent among the primary diseases of the lids from which gangrene may develop. *Grossmann* reports a case in which a pustule as large as a pea developed on the skin of the upper lid of a broommaker, from which a brawny oedema extended even

to the thorax, and was accompanied by a high fever. By the third day the skin of the entire lid had been transformed into a black crust. The patient recovered, but with an extreme degree of ectropion.

[Contusions and lacerations predispose to gangrene of the lids. In measles, scarlet fever, impetigo, eczema, and variella, it is usually caused by a secondary infection with streptococci, staphylococci, diphtheria bacilli, or germs of putrefaction. Gangrenous stomatitis, noma, may be transferred to the lids by the fingers.

It should be remembered that the application of **ice** to the lids may cause the skin to gangrene. For this reason ice should never be applied, but, when cold is needed, ice compresses, made by allowing strips of cloth or gauze to freeze to the surface of a cake of ice, should be used.

Treatment

Little can be done locally. Antiseptic dressings or soothing ointments should be applied and the patient given absolute rest. The main reliance must be placed on the general treatment and the casting off of the sloughs awaited. When due to anthrax intravenous and local injections of anthrax serum should be added.]



Fig. 6
Herpes facialis



1

2

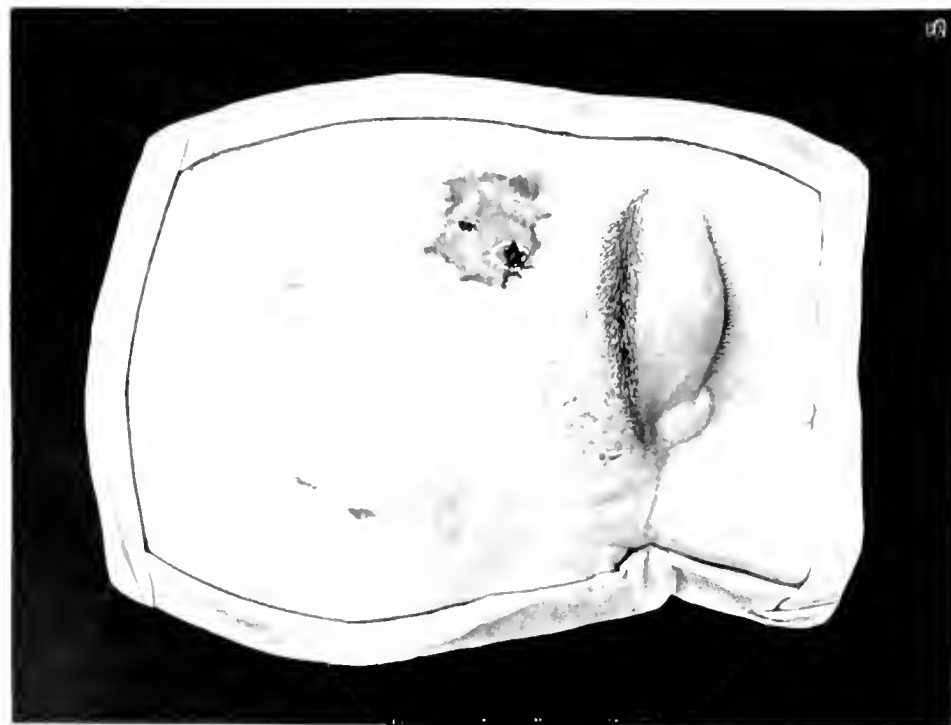


Fig. 7
Herpes zoster ophthalmicus



Fig. 8.
Herpes zoster gangraenosus.

Herpes Zoster Facialis.

Plate VI, FIG. 6; Plate VII, FIGS. 7 AND 8

Herpes zoster, shingles, is an acute, infectious, exanthematous disease characterized by fever, prostration, and an eruption of vesicles in the area of skin supplied by a certain nerve. One attack usually renders a patient thereafter immune. The vesicles vary greatly in number in different cases, contain a fluid that is clear at first but soon becomes turbid and purulent, and finally burst, creating ulcers over which crusts are formed. These ulcers leave permanent scars. The disease is met with chiefly in the spring and fall. [The site of the primary lesion has not been positively ascertained, but is probably in the spinal ganglia. The nature of the morbid agent is likewise uncertain as yet.]

The *trigeminus* is the cranial nerve most often affected. Each of its branches may suffer, but the first branch is much more frequently involved than the others, and the disease is then known as **herpes zoster ophthalmicus**, or *zona ophthalmica*. An inflammation is then present either in the Gasserian ganglion, the ciliary ganglion, or the peripheral portion of the nerve. [Its onset is marked by severe neuralgic pain, which may persist long after the eruption has subsided. The skin supplied by the nerve becomes more sensitive and reddens, while the eyelid becomes oedematous, sometimes to such an extent that the eye can hardly be opened.] Then the eruption appears on the upper lid, the forehead up to the edge of the hair, and the nose, almost always sharply limited by the median line of the face (see Plate VII, Fig. 7). Fig. 8, Plate VII, taken from *Jacobi's "Dermochromes,"* shows a case of herpes zoster gangraenosus in which the ruptured pustules covered with crusts were unusually numerous and penetrated deeply into the corium.

Complications on the part of the eye

[Vesicles frequently appear on the conjunctiva, and the cornea is involved in about one-third of the cases. Vesicles sometimes develop

¹New Edition just issued. Text by Jerome Kingsbury, M.D. and W. G. States, M.D., Rebman Company, New York.

on its surface and form deep ulcers. In other cases deep infiltrates appear in the cornea and persist for a long time. In still others the surface may appear stippled, or may show a cloudiness in which no defects in the epithelium can be seen. A neuroparalytic keratitis sometimes develops later as a result of paresis of the trigeminus.

Other complications sometimes seen are iritis, either primary or secondary to a keratitis, a subacute scleritis, a paresis of some of the extrinsic muscles of the eye, and an optic neuritis, which may result in atrophy.]

When the second branch of the trigeminus is affected the eruption appears on the lower lid and over the superior maxillary and malar bones (see Plate VI, Fig. 6).

[The duration of the cutaneous eruption is ten or twelve days, but the inflammation of the eye may last for several weeks. The skin is usually hyperaesthetic during the inflammation, but areas of anaesthesia may appear later. Considerable disfigurement follows the gangrenous form, and even in less severe cases the skin may be given a mottled appearance by the scars that are left.]

Diagnosis

The diagnosis is easily made from the neuralgic pains, the confinement of the eruption to the area supplied by the nerve, and the fever. In almost every case herpes zoster ophthalmicus is confined to one side of the face, although it may be bilateral at times, as was pointed out by *Hutchinson*. It can be differentiated from simple herpes facialis (labialis) by the vesicles themselves; in simple herpes the epidermis alone is elevated, while in herpes zoster the ulcer sinks deeply into the corium and leaves a scar.

Although the cicatrices may cause more or less annoyance the prognosis is usually good, [unless the eye is seriously involved, when the sight may be impaired or even lost. The disease is sometimes fatal in old and feeble persons.

Treatment

The indications for treatment are to relieve the heat and irritation of the affected skin, to protect the eye, to ease the severe pain and to procure sleep.

The first indication may be met in three ways; the skin may be covered with compresses wet with a lead and opium wash, or with a boric acid solution; or it may be dusted with a powder, starch, rice, lycopodium, or talcum, to dry up the vesicles; or an ointment may

be applied containing zinc oxide, boric acid, xeroform, guaiacol, or collargol. Good results have been ascribed to ointments containing orthoform, anaesthetin, or acoin, covered with a bandage; these remedies produce at first a lively smarting, but this is followed by relief that lasts from four to six hours.

When the conjunctiva is affected it should be washed out with a saturated solution of boric acid two or three times a day. When the cornea is attacked the treatment for ulcerative keratitis should be instituted at once; bathing the eye with as hot water as can be borne for ten to twelve minutes every two or three hours, and instillations of atropine three times a day. The electric thermophore is a pleasanter method of applying heat when it is available. Subcutaneous injections of dionin, 0.01 to 0.02, are said to relieve the pain.

The pain can sometimes be relieved by passing the galvanic current through the trigeminus, the positive pole to be placed on the neck. Pyramidon has been recommended in doses of 2 to 3 grams a day. Quinine, phenacetin, acetanilide, and other similar drugs are occasionally efficacious, but very often relief from pain and sleep can be secured only with the aid of morphine.

The salicylates may be given internally, and the bowels should be kept open with mild laxatives.]

Variola Vaccina.

Plate VIII, FIGS. 9 AND 10

Cow-pox is occasionally seen on the lids, usually as the result of direct transmission from a vaccination on the arm or leg. Sometimes the child scratches the sore and then rubs the infection into the eye with the finger, sometimes the mother or the nurse infects the eye with her own soiled finger, or with a handkerchief that the child has used, [sometimes a physician carelessly brings the lymph into contact with his own eye.] **Prophylaxis** consists of simple measures to avoid such accidents.

The **stage of incubation** may last only 3 or 4 days. The eruption is usually found along the intermarginal space of the lid [because the epithelium there is very delicate, easily injured, and always moist. The margin of the lid begins to swell at a certain point]. One or more vesicles appear and change into pustules in a few days, while the signs of inflammation, chemosis and oedema, steadily increase. The pustules rupture, [but instead of drying up, as they do elsewhere on the body,] they form large, flat ulcers covered with a gray coat [as the result of being kept constantly moist. By this time the lids are oedematous and hard, and the amount of conjunctival secretion is greater than usual.] Recovery begins after 8 to 12 days and is complete in from 2 to 3 weeks. The lesion may also be located on the conjunctiva or cornea.

Diagnosis

The history ordinarily gives the clue to the trouble, but even when it does not, the diagnosis is not apt to be difficult. Differentiation from small-pox, in which the eruption is present elsewhere on the body and is attended with grave constitutional symptoms, is easy. A chancre does not, as a rule, cause as severe local inflammatory symptoms. It is possible to mistake it for a diphtheritic ulcer on the margin of the lid, but in diphtheria we usually find a whitish membrane covering the conjunctiva and the base of the ulcer looks dirty after the grayish coating has been removed, while in vaccina-



Fig. 9. Variola vaccina I.



Fig. 10. Variola vaccina II.

tion the ulcer has a clear red base and no membrane is present on the conjunctiva.

Prognosis

[The prognosis is almost always good. Sometimes a delicate gray disk, less dense at the center, is formed on its surface, occasionally surrounded by parallel rings, but ulcers are formed only in very severe cases and these alone leave permanent opacities. Serous iritis forms a complication in a few cases.]

Treatment

Treatment should usually be limited to keeping the eye clean and the application of some soothing unguent, like boric acid ointment. No irritating application should ever be made. When the cornea is affected the usual treatment for ulcerative keratitis, atropine and heat, should be adopted.

Chancre of the Eyelid.

Plate IX, FIG. 11

The **Hunterian chancre**, the primary lesion of syphilis, is occasionally seen on the eyelids of patients to whom the disease has been communicated by a person suffering from active syphilis either through the fingers, through kissing, [or through the removal of a foreign body from the eye. Physicians have been inoculated when treating the throats of syphilitic patients by having mucus coughed into their eyes.]

The lesion takes place almost invariably on the *intermarginal space* or the conjunctiva, because the more delicate tissue here affords less resistance than the skin to the entrance of the virus.

[After an incubation period of from 2 to 4 weeks] one or more hard nodules appear on the margin of the lid. The epithelium at the apex of the nodule falls off in a few days and this excoriation deepens to form an ulcer with sharp-cut, elevated, indurated margins and a dirty, sloughy base. [Toward the end of the first week the preauricular and submaxillary glands begin to enlarge, and sometimes become so swollen as to suggest mumps. The first of these glands to swell is the preauricular if the chancre is in the outer part of the lid, the submaxillary if the lesion is in the middle portion. About 2 weeks after the appearance of the nodule a slow, painless swelling of the lymphatic glands in the neck shows that the disease has started to become general, and the secondary symptoms of syphilis follow about a month later.]

As the chancre enlarges it encroaches on the conjunctiva and may involve the cornea; the ulcer becomes infected with pyogenic organisms, and the conjunctival discharge becomes considerable. According to *Treacher Collins* a chancre of the lid is often followed by an interstitial keratitis of the same eye.]

Diagnosis

The diagnosis of a chancre of the lid is not easy. Before ulceration commences it may readily be mistaken for a hordeolum, or a



Fig. 11.
Ulcus durum palpebrae. Syphilite. Primary affection.

chalazion; afterward it needs to be differentiated from a vaccine pustule, [a chancreoid,] lupus, tuberculosis, and a diphtheritic ulcer. [During this early stage the only positive clinical characteristics it presents are its peculiar hardness and the very indolent swelling of the lymphatic glands, especially of the preauricula; but these may be simulated by sores to which irritating applications have been made. Neither *Wassermann's* test, nor *Noguchi's* luetin test are of much value at this time, because in neither does the reaction become positive until after the infection has become generalized. A positive reaction proves that the patient is already syphilitic and therefore lessens the probability that the lesion present is a chancre, while it renders the diagnosis of gumma more probable.

The only way in which a positive diagnosis of chancre can be made before the signs of general infection appear, is by the detection of the ***spirochaeta pallida*** in scrapings from the ulcer. The examination for this microorganism is more difficult than that for many others, so it is well that it should be entrusted to skillful hands at present, but it should be made in every doubtful case in order that valuable time may not be lost before commencing treatment.

Treatment

If the diagnosis is made by the discovery of the spirochaetae before the glands have become involved, an attempt should be made to abort the disease by the excision of the chancre. This measure has not met with success in the past, but it must be remembered that it was then necessarily attempted after the infection had become generalized, as the diagnosis was impossible until that time. If the focus of infecting organisms can be removed before they have an opportunity to multiply and spread, we can hope for an immediate and complete cure.

Another method of abortive treatment that has been recommended, is to destroy the spirochaetae by irradiation of the chancre with radium, but very little is known as yet concerning its results.

The abortive treatment must not be relied upon, as statistics have shown that it fails too often. A vigorous ***antisyphilitic treatment*** should be instituted. At present it seems wise to give an intravenous injection of a large dose of salvarsan, or neosalvarsan, if there are no contraindications, and then to commence the regular administration of mercury. Twenty or thirty intramuscular injections of the salicylate of mercury should be given in the first six months, at intervals of 5 or 10 days. The dose and the frequency of the injections

varies with the age, sex, weight and condition of the patient; it is well to begin with half a grain and feel our way up, so as to test the susceptibility of the patient and to determine the efficient dose in the particular case. This rule should be observed when mercury is given in any form. At the end of six months treatment may be discontinued for a month or two. *Wassermann's* test should be made at rather frequent intervals, and should it persist in being negative and if no secondary symptoms have appeared, it may be assumed as probable that the attempt to abort the disease has been successful. Still the patient should be kept under observation and treatment recommenced if the reaction at any time becomes positive.

If the *Wassermann* test is positive, or if secondary symptoms have appeared, the treatment for syphilis should be persevered in for about three years, and an attempt made to render the reaction permanently negative.

If not excised the chancre should simply be kept clean and dusted with calomel.]



Fig. 12 Xanthelasma.

Xanthelasma.

Plate X, FIG. 12

Xanthelasma, or xanthoma, is a benign neoplasm of the skin of which two forms are known, the planum and the tuberosum; the former alone is met with on the eyelids.

In xanthelasma planum a number of flat, straw-colored, or sulphur yellow, soft tumors, elevated slightly above the level of the surrounding skin, appear on the upper and lower lids about the inner canthus, often arranged symmetrically on the two sides of the face. [Occasionally they are to be found scattered about the entire eye, and, though usually multiple, solitary tumors are sometimes seen. Each growth is separated from the others as a rule by normal skin.] They grow very slowly and cause no pain or other trouble, aside from the disfigurement they may occasion. They occur chiefly in elderly people, and are met with most commonly in women who have passed the menopause. [Their cause is unknown, but a history of jaundice, or of other signs of liver trouble, is common.]

The tumor consists of nests of so-called xanthom cells, or enlarged connective tissue cells, which are filled with fat granules. Each nest is separated from the others by walls of connective tissue. Giant cells are sometimes found in them.

Treatment

These tumors are absolutely benign and need to be removed only for cosmetic reasons. [They are not apt to recur after excision, which is easily performed. Small tumors may be removed by the application of trichloroacetic acid. *Darier* states that radium, applied for from 10 to 20 minutes over each tumor, should bring about progressive absorption and leave no visible trace of its action. The same result is said to be obtained by the high frequency current applied with a special electrode.]

Retention Cyst of the Margin of the Eyelid.

Plate XI, FIG. 13

Retention cysts at the margin of the lid originate from occlusion of the ducts of either *Zeiss'* sebaceous glands, or of *Moll's* sweat glands.

A **sebaceous cyst**, [commonly called a wen when situated elsewhere, as on the scalp,] is a retention cyst of *Zeiss'* glands, and contains a sebaceous, whitish mass, composed of horny and degenerated epithelial cells, granules of fat, and cholesterin, within a thin sac of connective tissue. These cysts are apt to be multiple [and to form little, spherical, yellowish swellings about as large as millet seeds in the skin] at the edge of the lid.

When *Moll's* glands, which open into the follicles of the eyelashes, have their ducts occluded, little translucent swellings, varying in size from that of a pea to that of a cherry, are formed along the margin of the lid. [Those that are due to distention of the tubules are multi-locular and lined by a single layer of cells covered by a layer of striated muscle fibers; those in which the duct alone is distended are unilocular and lined by a double row of cells. They contain a clear fluid of about the same chemical composition as the perspiration.]

Diagnosis

The diagnosis is easy from the location, form and painlessness of the cysts and is rendered positive by an examination of the contents. Congenital dermoids occur beneath the skin of the upper lid at one canthus or the other, not at its margin as a rule, [but always at points where sutures and fissures existed during fetal life.] They form smooth, rounded tumors of variable size, over which the skin is freely movable, often grow rapidly at puberty and sometimes extend deeply into the orbit without displacing the eyeball. [A chalazion is always attached to the tarsus.]

Treatment

The only treatment of these cysts is extirpation, which must be thorough, as otherwise they are apt to recur.



Fig. 13.
Atheromycyste on lid mm.



Fig. 14.
Molluscum contagiosum.

Molluscum Contagiosum.

Plate XI, FIG. 14

Molluscum contagiosum is a smooth, semi-globular tumor of the skin, of a yellowish white, pinkish, or mother-of-pearl color, varying in size from that of a hemp seed to that of a pea, and with a central depression from which a part of its contents exudes when pressure is made upon it.

Its **contents** are soft, whitish, sebaceous, or semifluid, and contain numerous oval, very refractive bodies, the so-called molluscum corpuscles, which can be seen very well in sections of the tumor. *Virchow*, *Caspary*, and *Lesser* took these to be vesicular formations produced by a change in the cell protoplasm. *Bollinger* thought them one-celled parasites of the genus *gregarina*, while *Neisser* believed them to be epithelial cells filled with *gregarinae*. *Crocker* considered them like the *coccidium oviforme* described by *Leukhardt*. But the recent studies of *Muetze*, under the guidance of *Uhthoff* and *Axenfeld*, show that all of the transitions from normal epithelium to molluscum corpuscles may be observed, and that therefore these bodies are not protozoic, but are the degeneration products of epithelial cells.

The disease is **contagious**. If a growth appears on the margin of the upper lid, another soon develops on the lower lid at the point of contact, [and very often smaller, more recent lesions can be seen about an older one.] I once saw a blind person who had several hundred mollusca. *Retzius* was the first to prove them to be contagious by successful inoculation. The pathogenic agent has not yet been discovered.

These tumors are found on all parts of the body in persons of uncleanly habits, but chiefly on the genitals of adults and the eyelids of children belonging to the poorer classes. They are rarely single, but generally multiple along the margins of the lids. [They grow very slowly, and are apt to suppurate and slough, if let alone, leaving cicatrices that are seldom noticeable.]

Diagnosis

The diagnosis is made easily from the form of the tumor, with its central depression, and a microscopic examination of its contents.

Treatment

The best treatment is to snip away the tumors with scissors. [They may be opened and cauterized with nitrate of silver, trichloroacetic acid, or the actual cautery. In many cases a thorough evacuation of the contents with a pair of forceps is sufficient. According to *Berry* the wall of the cyst may be drawn out with a fine pair of forceps.]

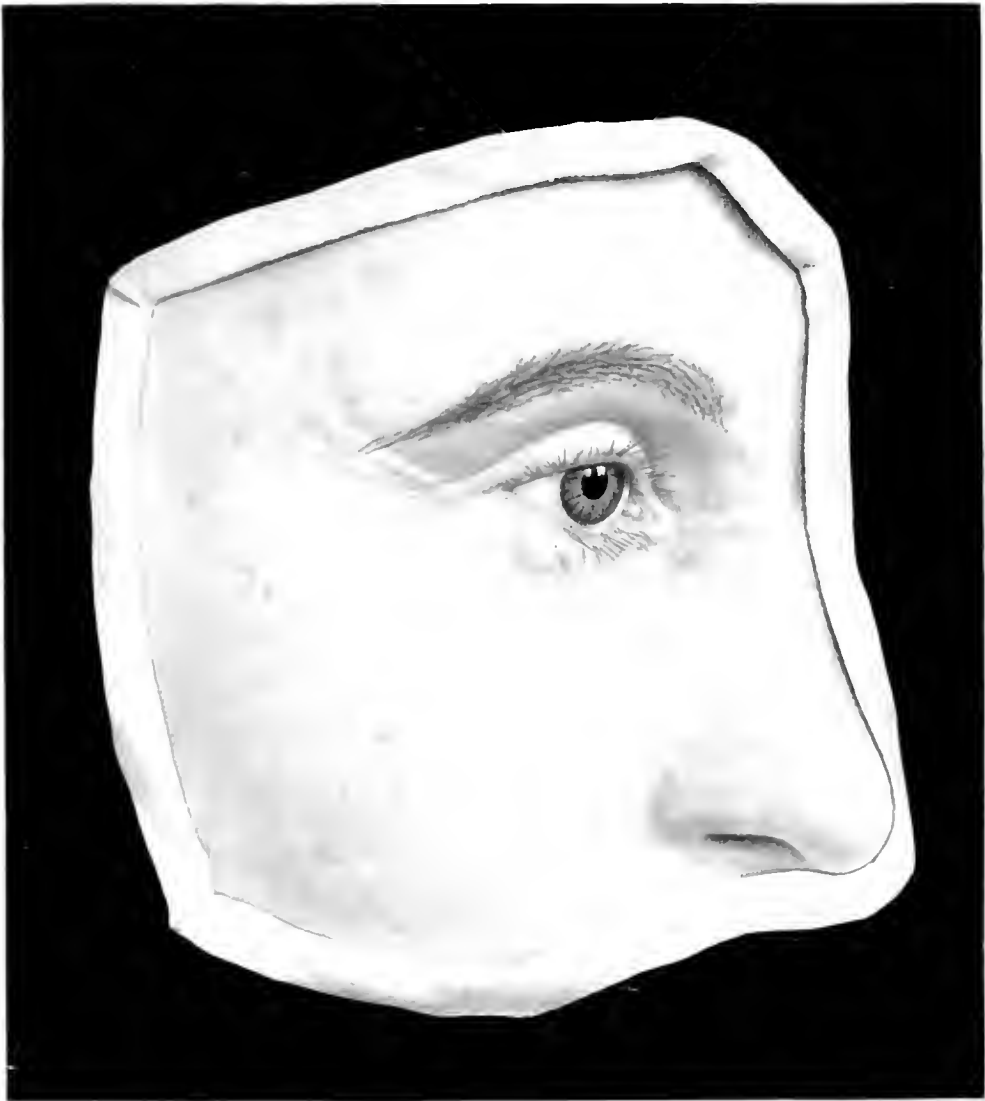


Fig. 15.
Hordeolum

Hordeolum.

Plate XII, FIG. 15

Hordeolum, or sty, is an acute, purulent inflammation of a sebaceous gland in the margin of the lid, and is therefore one of the forms of acne, although accompanied by more severe pain and greater inflammation than acne elsewhere on the body. This is due to the anatomical structure of the site of the lesion. [Two varieties are met with, the externum and the internum, which present nearly the same clinical picture.

Hordeolum externum, the more common of the two, is a staphylococcic infection of one of Zeiss' glands.] The first symptom is a diffuse redness and oedema of the lid with a painful feeling of tension. By careful palpation a small, hard, exquisitely tender nodule can usually be detected near the free margin of the lid, either between, or in front of the eyelashes, corresponding to the position of the inflamed gland. As the infiltration extends into the adjacent tissue the nodule increases in size, sometimes until it is larger than a pea, the skin over it becomes very red, and the pain becomes severe. After a few days suppuration begins in the center of the infiltrate, which soon breaks down, and a yellow point indicates where the pus will break through the skin. Perforation finally takes place, the pus is evacuated, and healing follows within a few days.

[More rarely a similar staphylococcal inflammation occurs in a Meibomian gland, which is a modified sebaceous gland, and forms a **hordeolum internum**. The primary symptoms are about the same as those just described, but the course is longer and more violent, because the Meibomian are larger than Zeiss' glands and are enclosed in the firm tissue of the tarsus, in which the hard, tender node can be felt by palpation. The conjunctiva as well as the lid is oedematous, the preauricular gland is enlarged and tender, the clinical picture is that of a deep-seated abscess of the lid. Perforation takes place through the conjunctiva as a rule.]

Prognosis

The prognosis is good. Recovery is rapid after the pus has been

evacuated, and the slight scar that is left is scarcely noticeable. [In rare and very severe cases a hordeolum internum may cause an extensive necrosis of the tarsus.]

Hordeolum occurs at all ages, but is most frequent between 12 and 25. Its development is favored by the presence of a slight chronic blepharitis, which gives the microorganisms present an opportunity to multiply enormously and to proliferate into and occlude the excretory ducts of the hair follicles, where the retained secretion readily undergoes suppuration with the assistance of the bacteria. [Only one hordeolum generally appears at a time, but others are apt to follow. Staphylococci are normally present on the margin of the lid, along with other microorganisms, and it is probable that they are often inoculated into the glands by rubbing the eye, particularly when the patient is suffering from a chronic blepharitis or conjunctivitis, but all cases do not seem to be susceptible of this explanation. A succession of sties is apt to be indicative of anæmia, chlorosis, or some other organic trouble which demands attention. It is very difficult to trace any connection between eyestrain and any of the inflammations of the margin of the lid, yet clinical experience seems to show that such a connection exists, because many obstinate cases of blepharitis and of successive sties quickly become amenable to treatment when glasses are worn that correct perhaps only a slight degree of astigmatism, or of some other refractive error.]

Diagnosis

The diagnosis is not always easy in the early stage. The violent onset, severe pain, and rapid swelling of the lid are quite suggestive of a serious disease of the eye, which is excluded when there is no abnormal conjunctival discharge, no circumcorneal injection, and the cornea is clear. When a very sensitive, inflamed infiltrate can be detected near the margin by passing the finger gently over the surface of the lid, the diagnosis is positive. In its later stages a hordeolum is readily recognized.

Treatment

Before suppuration has commenced an attempt may be made to abort the inflammation, but it generally fails. [A brisk purge should be given at once. The eye may be bathed with the hottest water it can bear without scalding for 10 or 12 minutes every 2 hours. When faithfully carried out this method proves more efficient than either

cold or hot compresses left on the eye for a considerable time, because after such a compress has remained on the eye a few minutes it is no longer cold or hot, but warm, and acts like a warm poultice to promote suppuration. If compresses are used they should be changed every 30 seconds for others either freshly torn from a cake of ice, or wrung dry out of hot water.

Other methods that have been recommended are to paint the margin of the lid with collodion, taking care that none drops in to eye, and to draw a mitigated stick of silver nitrate once across the tender spot. None of these methods do any harm, even when they do not succeed.]

When suppuration has evidently begun warm compresses are very grateful to the patient, as they lessen the pain by relieving the tension and swelling of the lid, while they hasten the suppuration. Such compresses may be made by wringing cloths or cotton out of a hot or warm 3% solution of boric acid, placing them on the eye for from 15 to 30 minutes several times a day, or keeping them on continuously, covered with rubber tissue which will keep them moist for from 6 to 12 hours, and changed often enough to prevent them from becoming dry. This is much better than to use chamomile tea, or any other of the ordinary household lotions, which are apt to contain impurities.

As soon as the presence of pus is certain it should be evacuated by an incision parallel to the margin of the lid and a moist dressing applied for 24 hours. If spontaneous rupture takes place we have simply to see that the cavity is well emptied, enlarging the aperture if necessary, and to apply a dressing. The symptoms then abate rapidly.

To **prevent subsequent attacks** we should first ascertain whether any chronic blepharitis or conjunctivitis is present and, if so, institute suitable treatment. The patient should avoid as far as possible causes of local irritation, such as impure air, tobacco smoke, and dust, for a considerable time, should cleanse the margins of his lids with a warm solution of borax, or of boric acid, each night before retiring, and should then rub gently into them a little yellow or white precipitate ointment. [When the blepharitis does not yield to this, the nature of the inflammation should be determined and the treatment modified accordingly. In some forms, such as eczema madidans, good results may be obtained by painting the margin of the lid daily with a 1% solution of silver nitrate.

Very often it is the general condition of the patient, rather than a local inflammation, that demands attention. Tonics are usually

needed, the digestive system often needs to be cared for. Exercise in the open air is frequently beneficial. It is also well to investigate the refraction and to correct any error that may be found, particularly if it is astigmatic, because relapses are less frequent when this is done.]

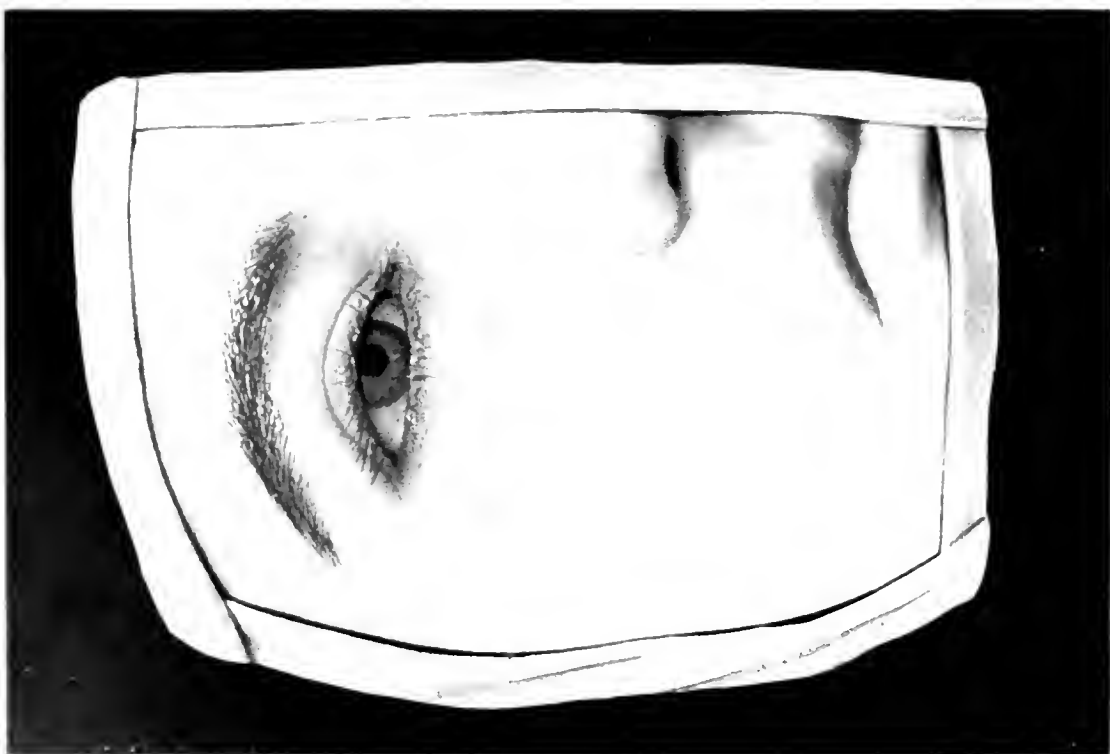


Fig. 16. Blepharitis marginalis sicca. Chalazion

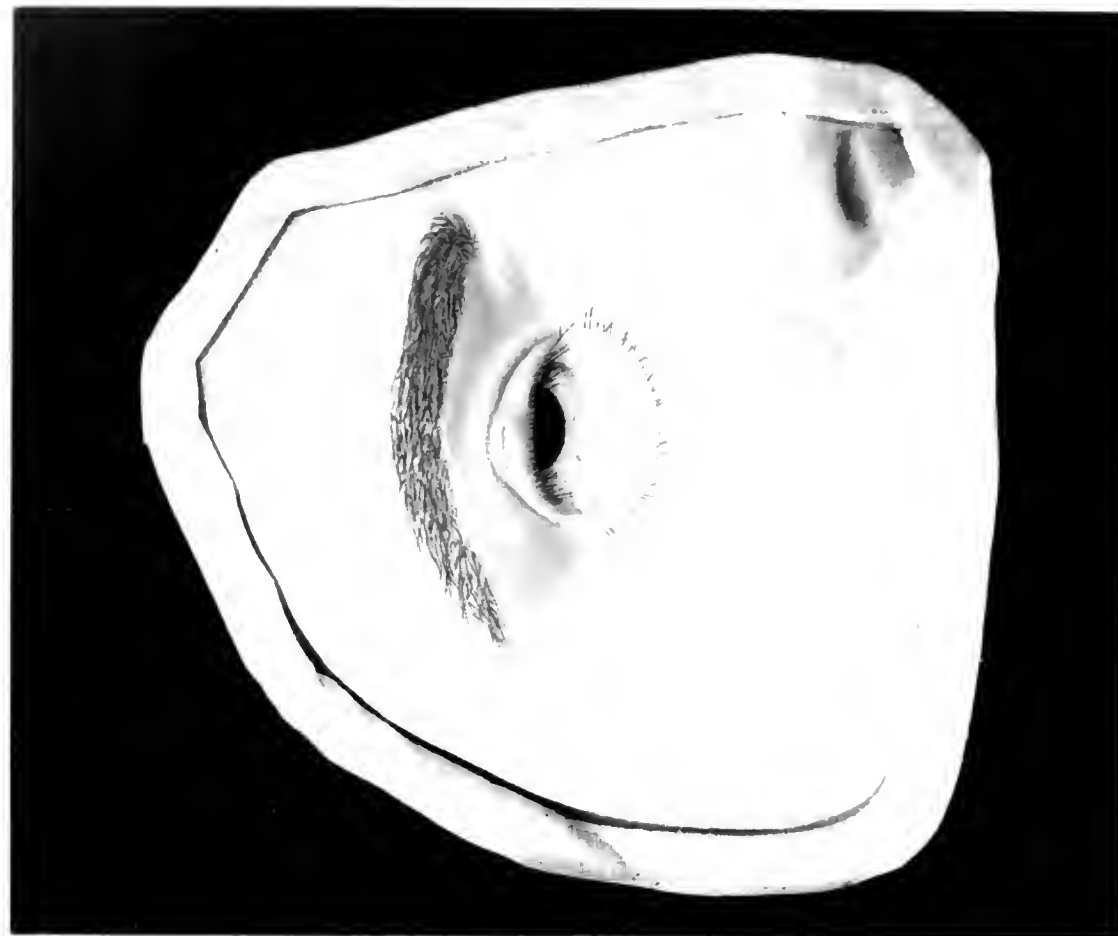


Fig. 17. Chalazion (seen from within)

Chalazion.

Plate XIII, FIGS. 16 AND 17

A **chalazion**, or Meibomian cyst, is a circumscribed tumor on the inner surface of the lid, beneath the conjunctiva, which originates in a Meibomian gland and is therefore attached to the tarsus. A Meibomian gland is simply a modified sebaceous gland, and is subject to pathological processes similar to those that affect the sebaceous glands of the skin, which it resembles in histological structure.

A chalazion is the expression of a **chronic disease** within the gland which progresses very slowly, presents almost no signs of inflammation, and may exist for years almost unchanged. In the course of some months a little nodule appears in the lid, but causes no trouble and is therefore not noticed by the patient as a rule until it is rather large. The skin of the lid is not reddened or changed in any way. When detected before it is large enough to form a prominence it feels on palpation like a minute, hard sphere beneath the skin, which is freely movable over it. In the course of time it becomes as large as a pea, a cherry pit, or a bean; then the lid is considerably deformed, especially if several are present. [Several adjoining glands may be affected simultaneously and give rise to a flat, broad swelling, which is called a giant chalazion.] The tumor is always movable with, but never upon the tarsus; this is explained by the manner of its development.

If we evert the lid, which may be difficult because of the mechanical impediment afforded by the tumor, we see a yellow brown or slate gray, rather translucent spot corresponding to the site of the enlarged Meibomian gland, surrounded by an areola of red and swollen conjunctiva. Finally the conjunctiva may be perforated, a rather thick slimy fluid flow away from time to time, and the tumor become a little smaller, but the main part, consisting of firm granulation tissue, remains unchanged in its capsule. [When the chalazion has thus ruptured the gelatinous-looking spot in the conjunctiva has been replaced by a small mass of granulation tissue.] Eventually, in the course of months or years, the mass of tissue in the capsule may shrink or undergo absorption until the tumor disappears.

Chalazia are apt to be multiple, and are more common in adults than in children, although they are met with at all ages. When small they cause little trouble, but when large they are annoying, not only because of the disfigurement they occasion, but also because of the mechanical impediment they afford to the movements of the lids and eye. Sometimes they become inflamed.

Cause

A predisposing cause in most cases is a slight, chronic conjunctivitis that leads to an occlusion of the excretory duct of the gland and the consequent retention of its secretion, [but a chalazion is not simply a retention cyst]. The retained contents of the gland thicken and may change into a hard, chalky mass from the deposit of lime salts, and so form what is known as a **calcareous infarct** of the Meibomian gland, or as lithiasis palpebralis. Such infarcts are to be seen as small white or yellow spots beneath the palpebral conjunctiva, are often found in considerable numbers about a chalazion, and it is quite probable that the inspissated mass of glandular secretion excites inflammation in the endothelium and adjacent tissues, which proliferate and become transformed into granulation tissue containing many round cells, as well as giant cells of an epithelioid character with many nuclei. The center of this granulation tumor has no nutrient vessels, so the tissue there is apt to undergo mucoid degeneration. [This accounts for the varying consistence and character of the contents of chalazia, which may be composed of a solid, thick, gelatinous substance, or of a transparent, or semi-opaque, curdy fluid, or of pus, when infection has taken place.] A dense, tough capsule is developed about the mass as the result of its pressure upon the surrounding tarsus, so that a chalazion consists of a granuloma enclosed in a fibrous connective tissue capsule.

A number of modern writers have claimed that chalazion is usually of a tuberculous nature, basing their belief mainly on the demonstration of numerous giant cells in the tissue, but this theory is disproved not only by the benign clinical course, [but also by the fact that the absence of tubercle bacilli has been demonstrated many times by inoculation experiments. The tendency of the disease to recur seems to indicate infection, but its morbid agent is still unknown.]

Treatment

Small chalazia sometimes disappear if we apply hot compresses and then massage them with an ointment containing yellow oxide of

mercury, ammoniated mercury, potassic iodide, or boric acid, or if we paint the lid with tincture of iodine, [or perhaps if we treat them with radium,] but such treatment often fails. The tumors should then be left alone if they are small, hard, and cause little or no disfigurement. If large and troublesome they need to be removed by operation.

Almost without exception the tumor should be approached **through the conjunctiva**, in spite of the fact that the protrusion is mostly outward. The natural opening of a chalazion is inward, the tumor masses are reached more easily, and less deformity is produced when the incision is made through the conjunctiva. [The rare cases in which the route through the skin is to be preferred, are chiefly those in which suppuration has taken place and the abscess is pointing outward.]

Anaesthesia is produced by the instillation of a 2 or 4% solution of cocaine into the conjunctival sac and the injection of a few drops of the same solution subcutaneously about the tumor. The lid is then everted, a chalazion forceps applied [with its plate lying on the skin, and its ring lying on the conjunctiva so as to enclose the chalazion in its fenestrum, and screwed down firmly. By this means the lid is held under perfect control and bleeding is prevented. An incision is then made parallel to the margin of the lid, down into the tumor, the contents and sac of which are then to be curetted away thoroughly with a sharp spoon.] An attempt may be made to remove the tumor entire by dissecting it out with scissors and forceps, [but such an attempt is not worth the loss of time and energy it entails; the operation is very difficult, because the capsule is apt to be firmly attached to the surrounding tissues, it usually fails and when it is successful nothing, so far as results are concerned, has been gained over the simple incision and curetting, while the patient has been subjected for a needlessly long time to a procedure that is at best annoying and unpleasant. The only cases in which it should be considered are those in which the chalazion is unusually large and hard. Clinical experience does not seem to bear out the theoretical danger of infection of the surrounding tissues when the capsule of a chalazion has been ruptured. After the last trace of capsule and contents has been expelled with the curette the lid clamp is removed, the lid replaced in its proper position, and the hemorrhage checked by cold compresses. Later a little sterile ointment may be applied to the margins of the lids.

In the rare cases in which a suppurating chalazion points ex-

ternally an incision may be made through the skin parallel to the margin of the lid and the pus evacuated. The entire sac and contents should be curetted out, and while this is being done the eye should be protected, either by the plate of a Desmarres' clamp, or the finger of the surgeon on the conjunctival side of the lid, as the conjunctiva may be wounded very readily.

Another method, seldom described though practiced to some extent, is for the surgeon to seize the chalazion between the thumb and forefinger of his left hand, one on the conjunctival surface of the lid, the other on the skin, make a deep incision into the tumor in the intermarginal space, at the opening of the duct of the gland, and then to curette out the sac and contents. The great advantage of this method is that the surgeon can feel any firm bits of tissue that may remain and so can control the operation by his sense of touch until he knows it to be done thoroughly, and that all traces of the chalazion have been removed. A disadvantage is that the hemorrhage has not a free exit and is apt to spread into the loose subcutaneous tissue of the lid and produce an ecchymosis, but this can be avoided to a great extent by bathing the eye with as hot water as can be borne at frequent intervals for a few hours. When this is done faithfully the resultant ecchymosis is frequently no greater than that produced by removal of the chalazion through the conjunctiva and the pressure of the chalazion forceps.]

To **avoid relapses** and the formation of new chalazia, any white calcareous infarcts that are to be seen should be cut down upon through the conjunctiva and removed with a small curette, and if a chronic conjunctivitis is present it must be treated and cured. [If relapses still occur the refraction should be corrected and then, if the case still prove obstinate, the advisability of a subcutaneous injection of staphylococcic vaccine should be considered.]

Blepharitis Marginalis.

Plate XIII, FIG. 16; *Plate XIV*, FIG. 18; *Plate XXVIII*, FIG. 41;
Plate XXIX, FIG. 42

Blepharitis marginalis is a generic term applied to a large number of inflammatory diseases of the margin of the lid. They are among the commonest of the diseases of the eye with which the general practitioner comes in contact, especially in large cities and among the anaemic and scrofulous children of the poor.

The skin of the eyelids is very thin and delicate and that at their margins is thinner and more sensitive than anywhere else on the surface of the body; therefore the margin of the lid is particularly apt to be affected in many skin diseases of the face, which require the same treatment as when found elsewhere, [modified only by the danger of irritating the eye]. It is also frequently the seat of certain characteristic forms of inflammation, of which the following deserve special consideration.

Hyperaemia of the margin of the lid, blepharitis vasomotoria, is not a true inflammation and needs to be differentiated. In some persons, usually blondes, the skin of the margin of the lid is so very sensitive that a slight external irritation, such as tobacco smoke, a sharp wind, or a bright light, or, in the absence of such an irritation, an unusual bodily exertion, a strain of the eyes, or the emotions, may suffice to cause them to become hideously red within a few hours. The eyes itch and burn, causing the patient to rub them and distracting his attention from his work, the lids feel heavy and hot, and by their drooping impart a more or less sleepy, tired expression to the countenance, while, because of the increase of the lachrymal secretion, the eyes look as though they were "swimming in tears." When we consider that the same condition of the eyes is brought about in most people by an all night carouse, with over-indulgence in alcoholic liquors, we can understand how vexatious it is for young persons with hypersensitive margins of the lids to present such an appearance. Not only is their enjoyment of innocent pleasures impaired by the disfigurement and the annoying irritation, but the symptoms are

often misinterpreted and have proved serious obstacles to success in business.

If the margins of the lids are observed closely during an acute attack they will be seen to be very red, and to present in the redness many minute, bright red, deeply injected blood vessels, often with a slight swelling of the lids, an injection of the palpebral conjunctiva, and an increase of the lachrymal secretion. As a rule no scales will be found on them or at the roots of the lashes.

When the trouble has been of long duration the frequently repeated acute attacks will have developed a chronic condition in which the margins of the lids remain red, thick, and heavy, with many large, distended vessels that vary in color from red to violet. The appearance created is very disagreeable and can be seen from a long distance. These patients suffer severely from photophobia and are accustomed to protect their eyes with great care. The trouble may be slight at first, but it is very persistent and often obstinate.

Treatment

Treatment should be begun at an early age and be both general and local. Many of the patients are anaemic or scrofulous and require careful attention in order to strengthen and harden their tissues. They must be warned against any excess or over-strain, late hours, too much reading, and living in bad air, and they must not be allowed to become effeminate. Life in the open air, healthful exercise and cold baths followed by massage of the entire body, will in time strengthen the organism and harden the delicate skin. Iron and quinine may be given internally as tonics.

[A careful examination should be made of the *refraction* of the eyes and any errors that may be found corrected with suitable glasses, for clinical experience has proved that the majority of these cases do well when this has been done. The refractive errors present in most cases are either hypermetropia, or hypermetropic astigmatism. The glasses to correct these errors must be worn all of the time, except during sleep; glasses worn only during study, or other near work, are not nearly as efficient, because the eye strain is then allowed to persist during a great part of the time.]

Local Treatment

This consists in bathing the margins of the lids with very mild astringents, such as cold lead water, an extremely weak solution of tannic acid, or water to which a few drops of alcohol or eau de

cologne have been added. Whatever solutions are used we must see that they are not strong enough to irritate the sensitive margins of the lids. [Cold compresses wet in these solutions laid upon the lids occasionally are very grateful to the patient and are beneficial.] It is a good plan to douche the eye every two or three days for from 3 to 6 minutes with a small, not too forcible stream of water containing some suitable astringent and hardening agent like alcohol, eau de Cologne, or borax, directed against the gently closed lids.

As a rule it is not best to use *ointments*, because they are generally too irritating, but if the skin of the lids shows a tendency to chap it may be covered with a thin layer of pure lanolin, [almond oil, or vaseline,] every evening before retiring.

In old and obstinate cases the margin of the lid may be painted with a 1% solution of silver nitrate, or it may be touched very superficially once or twice with the mitigated stick.

[In ***blepharitis angularis*** the margins of the lids at the canthi alone are red and sore, where the skin is moist and covered by a yellowish, viscid secretion. The symptoms are most marked at the internal canthus. This condition is due to a chronic inflammation of the conjunctiva induced by the Morax-Axenfeld diplobacillus, against which treatment must be directed. Our main reliance in this disease is sulphate of zinc. A drop or two of a $\frac{1}{3}\%$ to $\frac{1}{2}\%$ solution is instilled in the conjunctival sac two or three times a day, and a zinc ointment of the same strength is applied to the canthi at night. This disease is fairly obstinate and tends to recur if treatment is discontinued too soon, so the instillations should be kept up for at least six weeks after the symptoms have abated. In very obstinate cases in which this treatment failed, *Roemer* has succeeded in overcoming the disease by active immunization. He injects dead cultures subcutaneously in increasing quantities, maintaining the zinc therapy at the same time.]

The two principal types of inflammatory disease of the margin of the lid with which we have to deal are *blepharitis marginalis sicca*, and *eczema*.

Blepharitis marginalis sicca, known also as simple blepharitis, seborrhoea of the margin of the lid, blepharadenitis, and squamous blepharitis, is characterized by an increased secretion of inflammatory origin from the sebaceous glands, and is therefore in reality a **seborrhoea**. [When this secretion is very abundant the lashes seem to have been greased and the disease is called seborrhoea oleosa,] but in most cases the sebum dries into little scales that lie between

the lashes on the skin of the lid. Recent investigations have shown that these scales contain dried sebum and cast off epidermis, while beneath them may be found colonies of fungi on the margin of the lid and in the excretory ducts of the glands. It is probable that these fungi cause the disease in many cases.

The patient usually is led to consult a physician by a constant itching and burning at the margin of the lid, where little, if anything abnormal may be seen by a cursory glance. It is only on looking carefully, or after rubbing the lashes with the finger, that numberless minute, whitish gray scales, resembling dandruff, are to be seen at the roots of the lashes. After the lashes have been rubbed in this way the margin of the lid looks as if it were powdered with flour, and the scales may be dusted off by pulling and rubbing the lashes some more, [or they may be wiped off with a bit of wet cotton]. The skin beneath is then seen to be reddened, but not ulcerated. The cilia are apt to be loose and easily pulled out, but they usually grow in again unchanged; it is only when the disease has lasted a long time that they lose their luster, become bent and twisted, and finally fall out. Crusts that glue the lashes together are rarely formed; when they do form they will be found to be composed chiefly of dried sebum and to have no ulcers beneath them. [In acute conjunctivitis the lashes may be matted together by crusts of dried mucus or pus, as shown in Plate III, Fig. 3, but this does not necessarily indicate the presence of a blepharitis.]

Treatment

In treatment the important fact must be remembered that no ointment or other remedy will be of the least benefit unless the scales are all removed and the margin of the lid carefully cleansed before the application is made. [The scales may be removed by soaking them off with an alkaline solution, a solution of borax or of bicarbonate of soda, 1½ to 2%, a 5% solution of chloral, or with olive oil.] The best way is to rub a little pure olive oil between the lashes, allow it to remain a few minutes until the scales have become loosened, and then remove it by rubbing with a piece of flannel. [Another way is to make a lather of any good soap, rub it into the lashes as if about to shave, and then wash it off with a damp cloth.] Any masses that still adhere may be removed with cilia forceps, or some other instrument, until no traces of scales remain, [but this must be done very gently. The skin is very thin, delicate, hyperaemic, and easily wounded, the necessary manipulation causes a little, transitory swell-

ing and redness, and a slight wound is an annoying complication]. This cleansing must be repeated as often as new scales appear. No harm is done if some of the lashes come away during the cleansing; they would have fallen out sooner or later and they will grow in again as soon as the margin of the lid becomes healthy.

After the lid has been cleansed and dried a bit of ointment as large as a pea is placed on its margin and rubbed in, either with the finger or with a glass rod. As the procedure is rather irritating it is best to make these applications at first only once a day, in the evening before retiring, so that a thin layer of ointment will lie in contact with the skin during the night and can be washed off with soap and water in the morning. [As soon as it can be borne it is well to make the applications twice a day, but in the morning all excess of ointment should be very carefully wiped off that the skin may not have a greasy appearance.] The best ointment to use, as a rule, is Pagenstecher's, which contains 1 or 2% of yellow oxide of mercury. Hebra's diaehylon ointment, attenuated with an equal part of vaseline, is also useful. [Gradle has recommended zinc ointment. Sometimes all of these prove to be irritating and a 3% ointment of milk of sulphur and resorcin may be tried, or a simple boric acid ointment may prove sufficient.]

This disease has the reputation of being very obstinate, but it does not prove to be so when the treatment is carried out along the lines here laid down; it soon recovers, leaving no bad effects. [Failure is due almost every time to neglect in the preliminary cleansing of the margins of the lids.]

Eczema of the margin of the lid, also known as blepharitis eczematosa, ulcerosa, or serofulosa, psorophthalmia, lippitudo ulcerosa, and ophthalmia tarsi, presents a number of clinical pictures that correspond to the various stages of the disease, just the same as eczema on other parts of the body.

The first stage, **eczema papulosum**, is characterized by a hyperaemia of the margin of the lid associated with numerous little swellings, about as large as pin heads, produced by a serous saturation of the tissues with an immigration of leucocytes, which form small, red nodules.

As the serous saturation increases the epithelium is detached in places from the skin, and vesicles filled with a watery fluid are formed between the epithelium and the rete mucosum. This, the second stage, is known as **eczema vesiculosum**.

As the immigration of leucocytes gradually augments, the clear

contents of the vesicles become more and more turbid until it is finally purulent, and the disease has entered its third stage, ***eczema pustulosum***.

Finally the pustules rupture and leave erosions from which a serous fluid is constantly secreted. This fluid coagulates and forms crusts, beneath which the weeping erosions persist unchanged. In this stage the disease is known as ***eczema madidans***, or *rubrum*. New vesicles and pustules may form in its neighborhood, so that all four stages may be seen at the same time, the fourth predominating.

When the inflammatory symptoms subside the exudation lessens, the crust formation becomes not so great, and epithelium forms over the erosion, but for a considerable time it quickly becomes horny and is desquamated as whitish scales. This, the stage of healing, is called ***eczema squamosum***.

At the commencement of the disease the first three stages are apt to develop rapidly and to escape observation, but the fourth stage lasts for a long time and is therefore by far the most common and the one with which the physician is best acquainted.

In ***eczema madidans***, the stage of weeping erosion, the margin of the lid is much swollen, thickened, and covered with crusts that glue the lashes together. Pustules and vesicles can often be seen about the lashes near the crusts, and whenever one has ruptured a single cilium is to be seen rising out of a deep, crater-like ulcer, [presenting a clinical picture that resembles somewhat that produced by *sycosis coccogenes*]. These ulcers soon begin to weep, and then crusts form over them, the removal of which exposes a depressed surface that bleeds easily. If the disease is neglected the lashes fall out, the edge of the margin of the lid becomes eroded, and a slight entropion is produced. The eyelashes either never return, because their follicles have been destroyed by the suppuration, or return in a faulty position that is productive of the most serious consequences to the eye (see Plate XXII, Fig. 31).

In almost all cases the rest of the skin of the lid is involved, while usually the *eczema* extends to the cheek or the nose. [It is particularly common about the canthus, as its development in that situation is favored by a constant wetting. In obstinate, recurrent cases the skin of the lid may become so greatly thickened as to cause the disease to deserve the name of *pachydermatie eczema*, or *eczema callosum*.] The conjunctiva is almost always involved. Conditions that aggravate and maintain the trouble are often to be found in the nasal cavity.

Cause

The causes of this disease may be general or local. The general cause is almost always scrofula or tuberculosis. Most of the patients are children in whom we find all of the other signs of scrofula, such as swollen lymphatic glands, a bloated appearance, and thick lips. Less often local irritations created by bad air, dust, the conditions of daily work, or the secretions in cases of persistent conjunctivitis or epiphora, are sufficient to excite an eczema in persons who are not thus afflicted. [Maceration of the skin, at the canthus or elsewhere, favors the development and spread of the disease. Secondary infection is also quite apt to take place, and the danger of this is greatly increased by rubbing the lids to relieve the itching, which is very annoying.]

Diagnosis

The diagnosis is not difficult. Crusts are formed occasionally in simple blepharitis, but when these are removed the skin beneath is found intact, though hyperaemic, while in eczema it is deeply ulcerated. Sycosis develops almost invariably in adult males, while eczema of the margin of the lid usually affects weakly children. In the former the ulcers are usually discrete, though in bad cases they may occupy the entire margin of the lid, [but even then the absence of eczema elsewhere renders the differentiation probable. In any case of doubt the contents of the pustule and the diseased cilium should be examined with the microscope; sycosis parasitaria is caused by a trichophyton, sycosis coccogenes by a staphylococcus.]

The **course** of an eczema of the margin of the lid is very chronic if it is not properly treated. It passes from an acute into a chronic condition in which the epithelial covering of the skin becomes wholly necrotic and the denuded areas are covered by thick, yellow brown, firm crusts, a condition in which the disease may last for years.

As the result of a long continued eczematous blepharitis the **eye** suffers in various ways, but particularly from a chronic conjunctivitis, which may cause much trouble, and from the destruction of the cilia and of the edge of the margin of the lid. The hair follicles and bulbs are destroyed or injured by the ulceration and suppuration so that the lashes fall out and either do not grow again, or return as a few delicate, minute hairs. The quadrangular form of the margin of the lid is lost by the rounding off of its edge, and while some parts of it seem to be atrophied, others are hypertrophic and thick. In this way

trichiasis may be produced, in which the lashes that remain are bent inward and rub against the surface of the cornea.

Treatment

Treatment should be both constitutional and local. [When we have to deal with scrofulous children we have to adapt our measures to each individual case, for we have no specific means with which to treat this diathesis as such.] We should seek to improve their hygienic surroundings and see that they receive proper nutrition. When practicable a sojourn at the sea shore, or in the country, may be prescribed with benefit. [We must rely on air, light, movement, and nutrition as remedies, and the first three are best obtained by having the children play, lightly clothed, out of doors in the sunlight. The play should be active enough to create a hearty appetite and sound sleep at night, but must not be exhaustive in its effort. While it is well to see that the weakly child is not tired out by its more robust playmates, all coddling must be carefully avoided. Nutrition is to be promoted by a properly proportioned diet of proteids and carbohydrates which can be digested and assimilated. Too much of any one form of food is to be avoided. Candy, cake and pastry should be allowed only in limited quantities. If the child has been permitted too great an indulgence in "sweets," it may be well to exclude them from the diet for a while and later to admit them in small amounts. Tea, coffee and beer should be forbidden.

The bowels should be regulated; when habitual constipation is present, the treatment may be inaugurated by a dose of calomel. Cod liver oil, in doses of a teaspoonful to a dessert spoonful three times a day, still remains the most efficient remedy of its class, and is well borne. Iron is needed as a rule; the syrup of the iodide of iron in doses proportioned to the age, is a favorite prescription with many. Small doses of *Fowler's* solution are also beneficial.] The complications to be found in the nose in very many cases must be cared for, because until they are cured the disease is apt to recur in the eyelids after it has apparently been healed.

[The first point in **local treatment** is to prevent the patient from rubbing his eyes. Children cannot be expected to exercise the great amount of self-control required to bear the itching without an energetic effort to relieve it, so it is well to interpose some mechanical impediment. Short, pasteboard splints about the elbows are serviceable, as they do not interfere materially with play and at the same time render it impossible for the children to touch their eyes with their fingers.]

The *crusts should be removed carefully* with blunt forceps every day after they have been loosened with hot water, an alkaline solution, or olive oil, taking away at the same time any loose or distorted lashes. This should be done very carefully and gently, though one should not be frightened if bleeding occurs, as the ulcers bleed very easily and new crusts are formed over them. It is often advisable at first to touch the base of the ulcer gently with the point of a crayon of silver nitrate, [or to paint it with a 1% solution of the same drug.] Then an ointment that is not too irritating should be applied and rubbed gently, but thoroughly into the margin of the lid. It seems to be less important what drugs the ointment contains than that they are not too much concentrated. [At the same time it must be remembered that fats cannot be tolerated by some patients, while pastes that are borne well by them, perhaps, are irritant to others. On the whole the most useful ointment for this purpose contains from 1 to 2% of yellow oxide of mercury.] Hebra's diachylon ointment, applied, as he directed, on strips of gauze that overlap one another, is also useful; in order not to incommode the patient too much, the eyes may be treated alternately in this manner. [Prolonged massage with mercurial lanolin, turpeth mineral ointment, or with collargol ointment, as well as the application of the same ointments on gauze during the night, has been recommended. Lassar's paste, 3 parts each of salicylic acid and starch, 30 of zinc oxide, and 100 of vaseline, is sometimes effective.] *Schreiber* has had excellent results with a $\frac{1}{4}$ % ointment of silver nitrate. When the eyes are very sensitive it may be best to use a 1% resorcin ointment, [boric acid ointment, or simply to smear the edges of the lids with vaseline.]

Whatever is used, it is important that the ointment should be fresh and not rancid in the least. The dressings should be changed once a day at first, later twice a day.

Under this treatment the eczema usually passes from the stage of madidans to that of squamosum, and desquamation begins. Ointments should now be discarded and the skin should be dusted with talcum, starch, or lycopodium powder, under the influence of which healing soon takes place.

When an eczema proves very obstinate and ointments have failed, it is sometimes advisable to paint the diseased portions of skin daily either with tar, or with equal parts of tar and olive oil. [One drug that is efficient in the treatment of eczema elsewhere must **never** be used on the eyelids because of the irritation it excites in the eye. This drug is chrysarobin. *Wirtz* has treated stubborn cases success-

fully by ionic medication, the induction of drugs by electricity, using a $\frac{1}{2}\%$ solution of zinc sulphate. Special electrodes are needed for this form of treatment.

Finally the ***importance of a careful investigation of the refraction*** and of the constant wearing of glasses that correct any errors that may be present, cannot be insisted on too strongly in the treatment of all forms of blepharitis. The connection between refractive errors and eczema of the lid may be very indirect, but the latter recovers much more readily when the former have been corrected.] Much patience is needed on the part of both the physician and the patient, but with it and careful attention, even the worst cases of eczema of the margin of the lid may be cured.

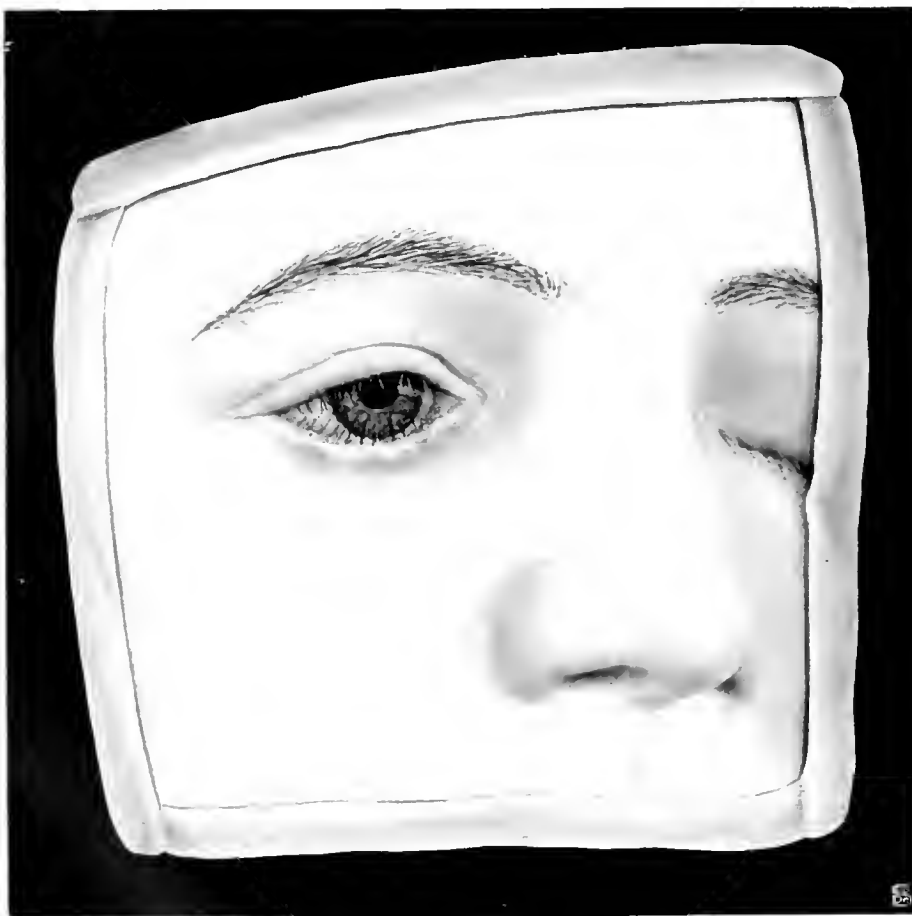


Fig. 18.
Entropium Trichiasis following
Conjunctivitis simplex chronica.

Entropium and Trichiasis.

Plate XIV, FIG. 18; Plate XXII, FIG. 31

By **entropium** is meant a turning in of the margin of the lid, so that the lashes come to lie upon the surface of the eyeball, even though they may be correct in position and arrangement as regards the lid; by **trichiasis** is meant a distortion in the direction and arrangement of the lashes, so that instead of forming a regular line and curving forward, they are irregular, deformed, and directed backward toward the eyeball, even though the margin of the lid may be in its proper position. The term **distichiasis** should be reserved for those cases in which the presence of a double row of lashes is congenital, because when this is the result of a pathological condition it is in reality an accidental form of trichiasis and should be known as such. Entropium and trichiasis often occur together, as shown in Figs. 18 and 31. In both conditions the eyelashes rub the surface of the eyeball with every movement of the lids, and act like foreign bodies to excite irritation and inflammation.

When an entropium is well marked the margin of the lid cannot be seen, as we look directly at the eye, unless we cause the lid to roll out by making traction on the skin with the finger; as soon as we let go it rolls in again and disappears. When the entire margin of the lid is inverted in this manner the entropium is said to be total, when only a portion turns in, it is partial. The middle and outer thirds of the lower lid and the outer third of the upper are the parts most apt to be affected in partial entropium. The two lids are about equally subject to this deformation.

Entropium varies in degree. In the mildest cases the margin of the lid is turned in just far enough to allow the tips of the lashes to rest on the eyeball. If the course of one of these lashes is traced it will be seen to have changed the plane of its curvature, so that instead of bending forward and outward it bends laterally and its tip is almost parallel to the margin of the lid as it slides up and down over the surface of the eyeball. As the margin of the lid turns in more and more, until the entire shaft of each lash is in contact with the eyeball, the curvature of the lashes gradually changes until they

bend in a direction exactly the opposite of normal, and follow the curvature of the surface of the eyeball with their tips directed away from the cornea toward the transition fold of the conjunctiva. In extreme entropium the skin of the lid lies upon the eyeball and the lashes lie deep in the conjunctival cul de sac.

The eye suffers severely from the mechanical rubbing of the lashes upon the sensitive conjunctiva and cornea. The patients feel at first as though they had foreign bodies in their eyes and suffer from pain, lachrymation and photophobia. In a short time the cornea particularly becomes affected, and it may be permanently injured. Its epithelium may become indurated, thickened, and opaque, as the result of the constant irritation or superficial ulcers may be produced by the infection of places where the epithelium has been scraped off by the lashes. In old cases the cornea is apt to be covered with thick, vascular opacities.

Entropium is divided etiologically into two varieties, the cicatricial and the spastic.

Cicatricial entropium is produced by the contraction of scars in the palpebral conjunctiva, which shortens the inner surface of the lid from above downward, draws inward and backward the margin, rounds off and flattens its inner edge, and so forces the lid to roll in. Such cicatrices are sometimes produced by wounds, burns, or pemphigus, but in the great majority of cases they are due to **trachoma** in its cicatricial stage (see Plate XX, Fig. 31).

Each trachoma granule leaves a scar when it disappears, and when many such granules have existed the remaining conjunctiva is filled with long, linear cicatrices, which run for the most part horizontally, and render it unable to cover the entire inner surface of the lid without tension. [Another condition produced by trachoma is a distortion of the tarsus, which is bent inward at an angle about 3 mm. above the free margin of the lid.] Trichiasis is added in almost all cases; the lashes are distorted, some fine and long, others short and stumpy, and scattered irregularly over the margin of the lid.

Treatment of cicatricial entropium

[The treatment of cicatricial entropium can hardly be divorced from that of trichiasis as the two conditions are so commonly found in unison and the indication in both is to do away with the irritation of the eye by the lashes. The simplest method of giving relief is by epilation, or pulling out of the lashes, and we have to resort to it frequently, although it is capable of only temporary benefit. Skill

and patience are often needed, especially when the offending lash is so small and colorless that it can hardly be seen with the aid of oblique illumination and a magnifying glass, and when seen evades the grasp of the forceps.

Electrolysis is the most satisfactory procedure when there are only a very few distorted hairs that touch the eyeball. A platinum needle attached to the negative pole of a battery is passed up beside the lash into the follicle, an electrode attached to the positive pole is placed on the temple of the patient and the circuit is closed. A minute froth appears about the needle which is left in place for a few seconds, the length of time proportioned to the strength of the current, and then withdrawn. If the bulb has been destroyed the lash may come away on the needle, or it may be pulled out easily, but if it remains firmly fixed in place the operation should be repeated.

All attempts to change the direction of the lashes by creating new tracks for them fail, because the life of a lash is only 150 days and even if it should be induced to take a new course its successor would not follow it, but would return to the original one.

Many procedures have had for their object the **destruction of all the roots** of the lashes. The ancient Egyptians cauterized the margins of the lids with red hot plates of gold, *Celsus* seared them with a red hot needle; in more modern times the skin has been turned back and the roots of the lashes have been excised or cauterized, and the entire margin of the lid has been cut away. Every operation of this nature leaves the lid in a badly deformed condition, and does away with the physiological activity of its margin, thus producing new evils to replace those caused by the scraping of the lashes. None of them are justifiable.

The **mechanical problem to be solved** in each individual case of cicatricial entropium is to replace and retain the margin of the lid in its normal position, in spite of the loss of its inner edge, the contraction of the cicatricial conjunctiva, and the deformation of the tarsus. In the solution of this problem the degree of entropium, the degree of trichiasis, the angle to which the tarsus is bent, and the condition of the cicatricial conjunctiva are factors that need to be taken into account. An entropium of mild degree implies a less degree of tension on the inner surface of the lid than an extreme entropium, and may be corrected by a traction which is readily overcome in the latter. When the in-turned lashes occupy the inner as well as the outer part of the margin of the lid an operation is apt to fail which may prove successful when they are confined to the outer

part. If the tarsus is badly deformed no operation in which this deformity is neglected can be expected to be permanently successful. In mild cases the question arises whether the condition is not progressive, for if the cicatricial contraction of the conjunctiva has not reached its maximum an operation may seem to be successful at first, and yet prove to be a failure. Hence the operation of choice in many cases is a combination of several methods, rather than any particular one.

When the entropium and trichiasis are slight, and the tarsus is not greatly bent, *Hotz'* operation is excellent. The skin continuous with the outer edge of the margin of the lid is drawn up and anchored to the tarsus so as to produce a permanent traction to counterbalance the tension on the inner surface of the lid. An incision is made along the upper margin of the tarsus from one canthus to the other, the skin dissected down to expose the orbicularis, and the surface of the tarsus is laid bare by the excision of a piece of this muscle 3 mm. wide for the entire length of the incision, care being taken to remove all of the muscle fibers. Four sutures are then introduced 2 mm. from the margin of the lower lip of the incision, passed upward through the aponeurosis on the upper third of the tarsus to points a little above the junction with the tarsoorbital fascia, and brought out through the skin of the upper lip of the wound. When tied these sutures secure the lower flap of skin tensely to the surface of the tarsus, which acts as an anchor to support the traction thus made on the margin of the lid. When needed this operation may be supplemented by making an incision along the conjunctival surface of the margin of the lid and filling the triangular gap thus formed with a graft taken from the skin behind the ear, or elsewhere, but an attempt to correct at the same time a distortion of the tarsus by the removal of a wedge-shaped section must weaken its holding power as an anchor. This operation is not suited for extreme degrees of entropium, or cases in which the tarsus is much distorted.

The **reconstruction of the margin of the lid** by means of grafts was suggested first by *Spencer Watson* in 1873, and since then all manner of variations have been made in the use of pedicled and non-pedicled flaps of skin and of mucous membrane for this purpose. The following operation should be credited perhaps to *Vossius*. Place a horn plate beneath the upper lid, and make an intermarginal incision 4 mm. deep from one canthus to the other, so as to split the lid into two laminae, the anterior one containing the lashes, the posterior the tarsus and the Meibomian glands. Dissect up the skin of

the lid obliquely for a distance of 5 mm. at each angle of the wound, then make a skin flap attached at both ends by two incisions about 4 mm. apart parallel to the margin of the lid, place this flap in the wound left by the intermarginal incision, secure it in place with fine sutures, and finally suture the wound in the skin.

One serious trouble with this class of operations is that unless the flap is taken from a portion of skin that contains absolutely no hair bulbs, the fine hairs which are normally to be found over most parts of the body are apt to continue to grow and to maintain the irritation of the eye which occasioned the operation. To avoid this danger grafts have been taken from the mucous membrane of the lip, and from the foreskin, when a simultaneous circumcision was feasible. Another danger in trichiasis is that some of the lashes will be included in the posterior lamina, or will grow either through, or on the inner side of the graft and so render the operation nugatory.

Whenever an incision is made through the tarsus for its whole length and traction is made on the outer edge of the margin of the lid a certain amount of **rotation** and it may be of **transplantation** of the margin upward is produced, but this was first made the object of an operation by the elder *Jaesche*. The best known of this class is the *Jaesche-Arlt* operation. An intermarginal incision 5 mm. deep is made so as to include the hair bulbs in the anterior lamina. A skin flap is marked out by two incisions, one parallel to and from 5 to 7 mm. from the margin of the lid, the other arching from one end of the first to the other, and excised without injury to the orbicularis. This skin wound is then closed with sutures which draw the anterior lamina with the lashes up on the anterior surface of the tarsus. No attempt is made in this operation to restore the margin of the lid, unless a graft is placed in the intermarginal incision, but the lashes are moved upward in the hope that in that position they will no longer be able to harm the eye.

Many operations have been devised in which the principal feature is the **correction of the distortion of the tarsus**. *Streatfeild* excised a wedge-shaped piece with its base outward from its entire length as nearly as possible at the place where it makes its angular bend inward. This has been modified in a great many ways. *Von Graefe* excised a triangular piece of skin with its base downward, then a triangular piece of the tarsus with its base upward, closed the skin wound with sutures, the middle one of which included the edges of the tarsus, and performed canthoplasty. Other surgeons excised larger or smaller pieces of the tarsus. About the beginning

of the nineteenth century *Saunders* removed the entire tarsus, an operation that has been revived recently by *Kuhnt*, with an improved technique, under the name of **enucleation of the tarsus**. The lid is everted, its margin drawn strongly upward, and a plate placed beneath its skin surface. An incision is then made parallel with and close to the margin of the lid through the conjunctiva and tarsus, perpendicular to the curve of the tarsus at that place. The orbicularis is separated from the anterior surface of the tarsus by blunt dissection to the convex margin of the latter, and then the conjunctiva is dissected off to the same point. The latter dissection is accomplished best with an instrument like a small periosteum elevator, a scalpel is not well fitted for the purpose. After the tarsus has been freed from all attachments except that of the levator to its convex margin, this is divided with scissors, and the conjunctiva may or may not need to be held in place by a three mattress sutures brought out through the skin of the lid. Ptosis is not produced by this operation, as might be feared, and the results are excellent, so far as the entropium is concerned. If trichiasis remains and lashes continue to irritate the eye although the margin of the lid is in good position, some secondary operation, like the implantation of a graft, may be necessary, but in the majority of cases the offending lashes are few in number and can be removed by electrolysis. Many of the patients are content with their periodic removal by epilation.]

Spastic entropium is brought about by the contraction of certain fibers of the orbicularis when they do not meet with the resistance afforded by a normally tense skin. The most powerful contraction of this muscle is unable to produce entropium when the eye is healthy and the skin normal, so we need to study the construction and action of the orbicularis in order to understand how it can produce such an effect when the skin of the lid is lax.

The **orbicularis** is a thin, broad, circular muscle lying just beneath and attached to the skin of the eyelids. It is divided into two portions, the *palpebral*, which encircles the palpebral fissure, and the *orbital*, which extends from the former to the neighborhood of the margin of the orbit. The palpebral portion suffices for all ordinary movements of the lids, the orbital portion draws together the skin about the eye and takes part in the forcible closure of the lids. The fibers of the palpebral portion have a double curve, one with its concavity toward the margin of the lid, the other with its concavity toward the eyeball and corresponding to its curvature. When these fibers contract both curves straighten, thus bringing the lids

together and pressing them upon the eyeball. As long as this nicely balanced effect is maintained entropium cannot be produced, but when the skin of the lid is abnormally lax the muscle fibers next to it do not receive their normal support, and the effect of this is that the force of the contraction of the fibers situated more internally preponderates and forces the margin of the lid to turn in.

Spastic entropium usually affects the lower lid and is met with most frequently in old people whose skin has lost its elasticity; hence it is often called senile entropium. An attack is frequently brought on in such people by the prolonged application of a bandage, and is therefore a not uncommon and very annoying complication after an operation for senile cataract.

The same fibers of the orbicularis likewise preponderate when their contraction does not meet with the counter-pressure normally supplied by the eyeball, as in enophthalmos, atrophy of the eyeball, or after enucleation. When the eyeball is absent we find almost always an entropium of both lids.

Sometimes an entropium of the lower lid is produced in children who are suffering from eczematous conjunctivitis and blepharospasm. In these cases a swelling of the transition fold of conjunctiva forces outward the lower part of the lid and its margin is pushed inward by the violent contraction of the orbital portion of the orbicularis which presses the margins of the two lids together.

Treatment

[The treatment of spastic entropium depends first on its cause. When it is due to blepharospasm in a case of eczematous conjunctivitis a cantholysis should be performed at once to stop the spasm and permit the disease to be treated. After an enucleation it is corrected by the insertion of an artificial eye. In senile entropium the treatment has to be determined by the degree to which the skin is relaxed.

Senile entropium induced by a bandage can often be cured by removing the dressing and drawing the lid down frequently with the finger. When this does not suffice the lid may be painted with collodion, which contracts as it dries and furnishes a temporary support, or strips of adhesive plaster may be applied from close to the lashes down upon the cheek, thus drawing the skin slightly downward and supporting it artificially. Many other mechanical appliances have been devised to draw the offending lashes away from the eye, but those mentioned are as good as any. Pinching the skin

of the lid frequently for a considerable time probably acts by stimulation, whether it is done with the fingers, as by *Sichel*, or with forceps, of which a number have been devised for the purpose since the first was invented by *Rhases*.

After such measures as these have proved ineffective operative treatment is to be considered. The purpose of this must be either to strengthen the skin as a support for the action of the orbicularis, or to weaken this muscle.

Excision of a horizontal fold of skin has been practiced since the days of *Celsus*, but alone it is seldom able to accomplish its purpose for any great length of time. The skin is so loose and so easily stretched that the removal of a portion does not render the balance sufficiently tense to act as a base of action for the muscle. *Skokalski* tried to obviate this by the production of a broad, subcutaneous cicatrix. He dissected up almost all of the skin of the lower lid so as to form a quadrilateral flap, cut away a portion of the lower part and then sutured the lower edge of the flap to the lower edge of the wound. His results are said to have been good, but as a rule it is best not to rely on the excision of a fold of skin, although such an excision is sometimes advisable as an adjunct to other procedures when the skin is redundant. Vertical strips of skin should never be excised; the results are no better and unsightly scars are left.

The application of **caustics**, and of the **actual cautery**, to the skin of the lids in order to produce a cicatricial contraction that will afford the needed resistance to the orbicularis, has been advocated repeatedly from the times of *Hippocrates* down to the present day, but the serious objection seems to be that it is difficult to so gauge the application of caustics as to produce the exact amount of effect required. *Theobald* says with regard to the use of caustic potash for this purpose: "Our aim should be to produce an eschar 3 to 4 mm. in width, parallel with the lid margin and extending the whole length of the tarsus. At no point should the action of the caustic be allowed to approach nearer the margin of the lid than $1\frac{1}{2}$ to 2 mm., and as the destruction of tissue tends to spread considerably beyond the actual point of contact of the crayon, the line of application of the latter should be about 4 mm. from the ciliary border. Along this line, the lid meanwhile being everted and kept well upon the stretch, the crayon should be drawn back and forth several times, until the epidermis is destroyed and the tissues begin to assume a brownish color. Holding the lid carefully so that it shall not become inverted, the action of the caustic is allowed to extend as far as may seem

desirable, when it should be arrested quickly by the application of an acid solution."

The method that is, perhaps, in most common use is to try to **increase the tension of the skin**, or to attach the edge of the lid to a more or less firm support, by means of the cicatrices produced by the introduction of ligatures. *Hippocrates'* operation is the oldest of this class and is efficient in many cases. Pass a thread through a fold of the skin and allow it to suppurate out. Sometimes it is better to insert two or three stitches in this manner, a little distance apart.

A neater operation is as follows: Take a suture armed with a needle at each end; introduce the needles a short distance apart 3 mm. from the margin of the lid, pass them down vertically beneath the skin, parallel with each other, for a distance of 15 mm. and bring them out, thus leaving a loop of thread lying on the skin near the lashes. Tie the ends of the suture over a roll of cotton tightly enough to produce some ectropium and leave it in place for several days.

Snellen's operation is to introduce both needles at the bottom of the transition fold, pass them directly out through the skin, leaving a loop of thread on the conjunctiva in the fornix lying parallel with the margin of the lid, reintroduce the needles through their wounds of exit, carry them up just beneath the skin almost to the lashes, bring them out about 2 mm. apart and tie them over a roll of cotton.

It must be remembered that *although all of these procedures are useful in spastic entropium, none of them are of avail in the cicatricial variety*, to which they are essentially unsuited. This warning seems to be needed because the operative procedures applicable to the two conditions have been confused so often in the past.

Interference with the action of the orbicularis was first proposed by *Key* in 1825, and since then various canthoplastic operations have been devised. The most generally applicable and efficient one of these is, perhaps, that devised by *Agnew* and called by him **cantholysis**. The tissues at the outer canthus are divided horizontally with a strong pair of straight scissors, the points of a smaller pair of scissors are passed vertically into the wound so that one blade shall be on each side of the palpebral ligament, which is then divided while the lid is held tense by the other hand of the surgeon. The cut edges of skin and conjunctiva are then united with sutures. This operation is useful not only for the relief of spastic entropium, but also as an adjunct in many cases of the cicatricial form.]

Ectropium.

Plate XV, FIG. 19

By **ectropium** is meant an eversion of the eyelid so that its conjunctiva no longer lies in apposition with the surface of the eyeball, but is more or less exposed to the air. It varies in degree from a condition in which the inner edge of the margin of the lid does not quite touch the eyeball to one in which the entire lid is turned inside out, and may be present in either lid, or in both, but the lower lid is the more commonly affected.

The eye soon becomes very much irritated in the presence of even the slightest degree of ectropium. As soon as the edge of the lid is drawn away from the eye the lachrymal punctum is turned outward, so that it is no longer immersed in the lachrymal lake, the natural conduction of the tears is done away with and epiphora is produced, that is, the tears overflow the margin of the lid and trickle down over the cheek. At the same time air is allowed access to the conjunctiva of the lid, which becomes swollen and hypertrophic and forces the lid out still farther, thus inaugurating a vicious circle in which each condition is aggravated by the other. When the ectropium becomes of high degree the cornea may be imperfectly covered when the eye is closed, and this may give rise to a serious keratitis e lagophthalmo.

Ectropium is divided into four varieties, according to its etiology, paralytic, senile, spastic, and cicatricial.

1. **Paralytic ectropium of the lower lid** results from the paresis of the orbicularis accompanying facial paresis, and is brought about in the following manner: When the fibers of the orbicularis are paretic that muscle is no longer able to press the lid closely against the eyeball, but allows it to obey the law of gravitation and sink a little away from the eye, and thus mechanically to produce an ectropium.

2. **Senile ectropium** resembles the paralytic closely, but is not due to a muscular paresis. The orbicularis is weak and the skin relaxed as the result of senile changes, so that they cannot hold the lid properly against the eyeball, but allow it to sink downward and outward so as to expose the conjunctiva.



Fig. 19.
Lupus vulgaris faciei. Narbenectropium.

3. **Spastic ectropium** is met with chiefly in children and young people who are suffering from an acute conjunctivitis with much swelling of the tissues and a blepharospasm. The disease is usually an eczematous or scrofulous conjunctivitis, less often an ophthalmia neonatorum. It frequently happens when we try to open the eyes of such children that a strong contraction of the orbital portion of the orbicularis everts the lids, usually both together [or the upper one alone, less often the lower one only]. Sometimes this may be produced by a simple pressure on the lids, without touching the eye. In every case the combination of a swollen conjunctiva and a spasm of the orbital portion of the orbicularis is necessary to the production of a spastic ectropium. [Once everted the lids are held in that position by a contraction of the palpebral portion of the muscle,] so unless the eversion is corrected quickly the already swollen conjunctiva will become strangulated and oedematous, and the eyeball may be totally hidden beneath the swollen, everted lids. The ectropium will then be permanent until the conjunctivitis has been cured.

A slighter degree of ectropium may be produced by the simple swelling of the conjunctiva, which becomes not only thicker but broader, and forms a sausage like swelling that forces the margin of the lid away from the eyeball. This is known as a **mechanical** ectropium. The eversion will then be favored by a constricting or spasmodic contraction of the palpebral fibers of the orbicularis.

4. **Cicatricial ectropium** is the worst variety and is caused by the contraction of a scar that has replaced a loss of a portion of the skin of the lid. Ectropium is particularly apt to develop after burns of the skin of the lid and cheek, wounds of the lid, operations on the cheek, [lupus, syphilis, anthrax, or gangrene of the lids,] and caries of the margin of the orbit; the resultant cicatrix draws the margin of the lid more and more downward or upward, until finally the entire, very red and thick conjunctiva of the lid may be turned completely outward and nothing can be seen of the lid except its margin, which is far distant from the eyeball.

Treatment

[An ectropium should be corrected at the earliest possible moment because of the severe irritation of the eye maintained by the continual exposure of the conjunctiva to the air, and of the vicious circle created by the swelling of the conjunctiva and the increase of the ectropium.]

In the early stage of a **paralytic** ectropium the principal treatment should be directed against the facial paresis, while the eye is bandaged at night to prevent desiccation of the cornea, for if the paresis yields to treatment and passes away, the lid will be made to assume its proper position by the renewed activity of the orbicularis. If the facial paresis does not improve, such an operation as is used in senile ectropium will be necessary.

The development of a **senile** ectropium can sometimes be prevented, when it is in its earliest stages, if the patient will be careful to wipe his eyes always with an upward movement. The chronic conjunctivitis must always be treated, and an application of the silver crayon is occasionally of good service when the palpebral conjunctiva is very thick, although the use of this remedy is highly objectionable in most diseases of the conjunctiva.

When the ectropium is too far advanced for this procedure to be of service, and yet is not very great, *Snellen's* operation, described under spastic ectropion, may suffice, but the result is apt not to be permanent.

A satisfactory result can be obtained in many cases of paralytic and senile ectropium by **tarsorrhaphy**, or shortening the palpebral fissure by uniting the margins of the lids for a certain distance. When this is done at the outer canthus the operation of *Fuchs* is the best. Both lids are split for the desired distance by intermarginal incisions; the outer flap on the upper lid and the inner one on the lower are removed, so as to leave raw surfaces on the outer side of the upper and the inner side of the lower lid, which are placed in apposition and secured there by sutures passed through the conjunctival flap of the upper lid, the skin flap of the lower one, and then tied over rolls of cotton. The effect of this operation is to elevate the lower lid, which may be drawn outward at the same time by making the incision in it a little the longer.

In other cases it is better to employ either *Szymanowski's* or *Kuhnt's* operation. In the former an intermarginal incision is made in the outer end of the lower lid for a distance determined by the amount of shortening required, a triangular flap of skin is excised at the outer canthus, the adjacent skin of the lid is undermined, then drawn over the raw surface so as to cover it, and secured in place with sutures.

In *Kuhnt's operation* a triangular piece of tarsus and conjunctiva, with its base at the margin, is excised from the middle of the lid. This causes a fold to be formed in the skin which gradually smooths

out if it is small, but as it is unsightly it is usually better to prevent it by continuing the intermarginal incision at the base of the excised portion to the outer canthus, to remove there a triangle of skin with its base a prolongation of the palpebral fissure, and to slide the superabundant skin over to fill in the defect. Under no circumstances, except when a malignant growth has to be removed, should a triangular piece be taken from the entire thickness of the lid.

When a case of spastic ectropium is seen early the eversion of the lids may be corrected and kept in place by adhesive plaster, or a pressure bandage, but *Agnew's* cantholysis is almost always needed. In obstinate cases *Snellen's* operation in addition is useful. Three heavy silk sutures are introduced at appropriate distances apart in the following manner: Take a suture armed with a needle at each end; pass one of them through the conjunctiva behind the lower border of the tarsus, carry it down beneath the skin for a distance of 2 cm., and then bring it out; pass the second needle along a course parallel to that of the first, 3 or 4 mm. away, and then tie the ends of the thread over a roll of cotton tightly enough to produce a slight entropium. Leave the sutures in place two or three weeks to create cicatrices that will hold the lid in place. The idea is to draw the lower border of the tarsus downward and forward so as to rotate its upper border inward and so bring the margin of the lid against the eyeball.

In **cicatricial** ectropium the scar that produces it may be slight and surrounded by healthy skin, or it may be of any extent and form, while the skin of the entire side of the face may have been replaced by cicatricial tissue. No one method of operation is applicable to all cases, but the ingenuity and patience of the surgeon are taxed to the utmost to so combine the various plastic methods as to meet the ever varying needs presented.

When the margin of the lid is held everted by a small, sharply defined scar, a subcutaneous division of the cicatrix that allows the lid to return to its proper position without tension may be sufficient, but there is considerable danger of recurrence, which may, perhaps, be avoided to some extent by a daily massage of the lid. Sometimes it is well to do in addition a *Szymanowski* or a *Kuhnt* operation, especially if the lid has become lengthened.

When the scar is small and confined to the lid it may be excised by three incisions that enclose it in the form of a triangle with its base parallel to the margin of the lid, as proposed by *Dieffenbach*. An incision is then made downward and outward from each end of

the base of this triangle, the flaps of skin on each side are dissected up, their edges brought together and sutured.

When the ectropium has been caused by an extensive loss of tissue, skin has to be brought from other localities to replace that of the lid. None but the most general rules can be laid down for guidance. First the lid must be freed from the cicatrix that binds it, and mobilized so that though destitute of skin it can be made to assume its proper position. In doing this it must be remembered that the margin of the lid must be spared, so that when replaced it may be able to perform its normal functions; that all cicatricial tissue must be removed, and all bleeding checked without ligatures before the flap of skin is applied. In most cases the first incision is made parallel to the margin of the lid at a distance of 3 or 4 mm., the cicatricial skin is dissected up and all cicatricial tissue is removed with forceps and scissors until the lid is freely movable. The defect left should have an even edge and be of a simple geometrical form so that a flap may be accurately applied. Then it is best as a rule to suture the eyelids together temporarily, in order to prevent movement as far as possible and so give the graft a better opportunity to adhere. All transplanted flaps contract; they should therefore when marked out be at least a third larger than the surfaces they are to cover.

The defect may be filled in by a pedicled or a non-pedicled flap of skin. The latter is usually known as a graft. The advantages of a **pedicled flap** over a graft are that it is more likely to adhere, and that its contraction is less. Its disadvantages are that, when taken from the neighboring skin of the face, it leaves an additional and undesirable scar; when taken from the arm by the Italian or *Tagliacozzi* method, it entails a considerable period of suffering on the part of the patient, whose arm is held immovably in contact with the face by some such apparatus as a plaster dressing until the flap has become adherent. The flap should consist of normal skin with a small amount of subcutaneous fat attached, should be of the same shape as the defect and one-third larger, and should have a pedicle of such size and so placed that the flap will receive sufficient nutrition through it in spite of the torsion to which it will be subjected. The flap should fit and perfectly fill the defect without tension, and then be fastened in place by sutures. The wound produced by the excision of the flap is then to be closed by undermining the edges of skin and drawing them together; but when the flap has been taken from the skin of the face near the eye the proximal edge must not

be undermined lest the lid be drawn outward. *Thiersch* grafts can be applied to any area still left uncovered.

In *Fricke's* operation a tongue-shaped flap is formed from the skin of the forehead, temple, or cheek, with its base at the end of the defect in the lid, dissected up leaving a pedicle at its base, and twisted around into position. This operation may be used to repair either the upper or the lower lid.

Dieffenbach's operation is useful mainly on the lower lid when the defect is in the form of a triangle with its base toward the margin of the lid. An incision is made prolonging the base of this triangle toward the temple; from its outer end another is made downward parallel to the outer side of the triangle, thus marking out a quadrilateral flap attached below, which is dissected up and moved over to cover the defect. The edges of the triangular wound left on its outer side are drawn partly together by vertical sutures and the remaining area is covered by a *Thiersch* graft.

When the defect is of a quadrilateral form it may be covered by sliding flaps of skin from various directions. The variations in detail are as innumerable as the conditions to be met with, but when they are in place the flaps must coapt perfectly and show no tendency to separate. The least tension, shown by a little gaping, is very harmful.

A *Wolfe* graft is the best to employ when the skin about the eye is cicatricial and cannot be used for a flap. This graft is cut in the same shape as the defect, and one-third larger, from some distant portion of the skin, usually that of the inner surface of the thigh or arm. All subcutaneous tissue is carefully removed and then it is placed in position with its edges tucked under those of the defect, which have been undermined. In bad cases these grafts have to be repaired and renewed, but satisfactory results can be obtained with care and patience.

The *Thiersch* graft is not as good as the *Wolfe* for this purpose, because the secondary contraction is greater, but it is useful in burns or wounds before an ectropium has developed, as well as in the treatment of the secondary defects produced by other plastic operations. The skin of the inner surface of the arm or thigh is put on the stretch, its superficial layer is shaved off with a sharp razor, thence transferred to the raw surface and carefully smoothed out over the area.]

Carcinoma of the Lids.

Plate XVI, FIG. 20; Plate XVII, FIG. 21

Carcinoma is the commonest malignant tumor of the lids. A tumor in the ordinary sense of the word is seldom present, but we see instead a shallow ulcer that scarcely rises above its surroundings, and has a slightly elevated, wall-like margin. This superficial, slowly spreading disease is often called a **rodent ulcer**, but its histological characteristics are those of a true superficial carcinoma.

This cancer is most apt to start either at the inner canthus, where the conjunctiva joins the skin, or in the skin of the lid. It is well known that carcinoma has a predilection for places where mucous membrane and skin meet, as at the margin of the lip, and the anus, and when it starts in the margin of the lid it is apt to be more malignant, and more rapidly and deeply destructive than when its place of origin is in the skin itself. In the latter situation it is more likely to be benign and to have the character of a rodent ulcer.

The left side of the face is affected more often than the right, the lower more frequently than the upper lid, and the usual **site** of the disease is at the inner canthus, particularly in the region of the lachrymal sac, as was pointed out first by *Valude*. When it starts at this place the cancer is apt to penetrate deeply and soon to destroy the lachrymal sac; it may then erode the lachrymal bone and invade the nasal cavity, or the tumor masses may be pressed through the nasolachrymal duct into the nose.

Fig. 20, Plate XVI, shows such a superficial cancer of the skin of the upper lid which has passed over the bridge of the nose and is about to invade the other side of the face. The growth of such a superficial cancer may be very slow and cover a period of from 20 to 30 years, as in the case of an old woman, of whom Dr. *Schulz-Zehden* kindly permitted a wax model to be made, which is pictured in Fig. 21, Plate XVII. The cancer, which started at the margin of the lid, had existed for many years, had destroyed the eyelids, the skin of the forehead and of the cheek, and the bony framework of the orbit, and had excavated a large cavity on the left side of the face, in the bottom of which the atrophic eyeball, with a still distinct

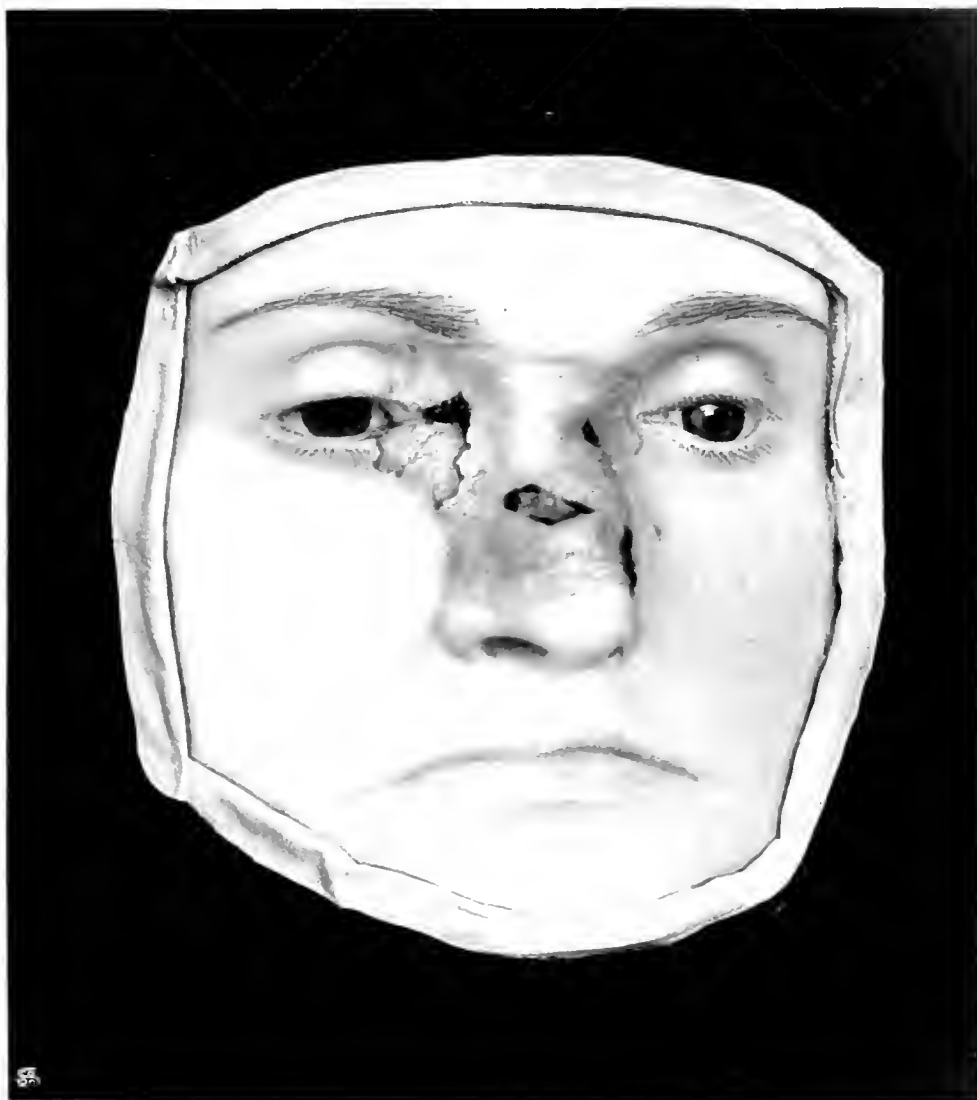


Fig. 20.
Carcinoma epitheliale I.

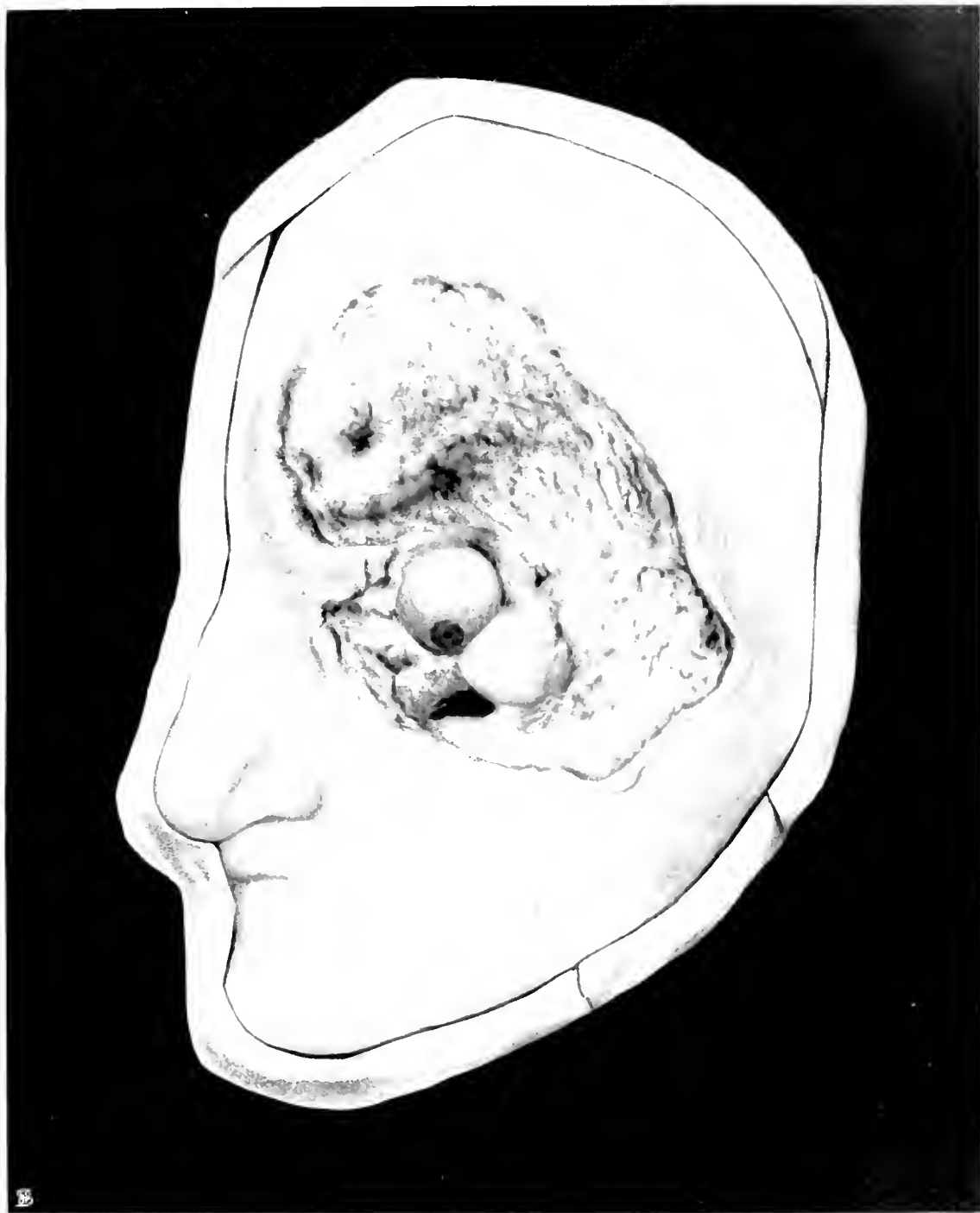


Fig. 21.
Carcinoma epitheliale palpebrarum II.

cornea, could be plainly seen lying far below its normal level. This patient steadfastly refused to consent to an operation, but kept the eroded area covered with a wet cloth up to the time of her death. The carcinoma was progressive, but so very slowly as to be remarkable.

Sometimes the carcinoma spreads by continuity in the skin of the nose and cheek, and sometimes it is not known until autopsy to what extent the neighboring organs and the accessory sinuses have become implicated.

As a general rule it may be said that this form of cancer *does not produce* secondary carcinomata of the lymphatic glands, or metastases in the internal organs. *Thiersch* found the lymphatic glands affected in only 2 cases; *Winiwarter* in only 2 out of 26 cases; *Mayeda* in only 8 out of 195, a percentage of 4.1.

Etiology

Little is known of its etiology except that cancer frequently develops from warts in this situation, from slight burns or abrasions, vesicles, nodules, and lupus.

Unna's description of the beginning of this disease is excellent. "Ulcus rodens starts as a rosy red or pearl gray nodule as large as a mustard seed, which rises a millimeter or less above the level of the surrounding surface. This enlarges laterally very slowly, while the center sinks in. Thus yellowish gray or reddish gray, smooth, oval spots are produced that vary in size from that of a pea to that of a quarter of a dollar, occupy the same level as the healthy skin, or may be a little depressed, are apparently, but not really, cicatricial, and are bounded by a delicate, ridge-like margin of mother of pearl color, in which there are often little nodular thickenings. No considerable thickening of the skin, or any peripheral inflammation, can be perceived anywhere. Even in this early stage a slight traumatism may produce an erosion in some part of its surface, usually in the center, which is covered by a dark crust composed of bloody serum and new epithelium. Left to itself this crust falls off and the lesion resumes its former appearance. Repeated erosions of the epithelium finally result in a permanent ulceration, which ushers in the second stage."

Treatment

[The benign nature of this form of cancer has led to many attempts to cure it by means of cauterization, or caustic pastes, either with

or without a preliminary curetting with a sharp spoon, but the results are not reliable. When a curettage is performed it must be very thorough, for if it is incomplete the condition will probably be aggravated. *Bulkley* says that *Marsden's* paste, made of equal parts of powdered arsenious acid and gum arabic wet with water, is efficient, and that a single thorough application is often enough to eradicate a small lesion, if left on long enough. Many other caustics, like zinc chloride and lactic acid, have been used for the same purpose, but a better method is irradiation with either radium or the x-rays, which are able to cure the disease, at least in certain cases, and leave scars that resemble the normal skin more closely than those usually left by other methods. *Judd* claims that 90% of the cases of epithelioma or superficial carcinoma may be relieved or cured by the **x-rays**, when there is no metastasis or glandular enlargement. In superficial growths he prefers a low tube, in deeper conditions a tube of more penetration, with short and frequent doses. Good results have also been reported from the use of the Finsen light.

Although enthusiastic claims are made for these means, and long courses of treatment urged when the response is not immediate, it hardly seems wise to waste too much time in such cases. When the response is not immediate it would seem better that excision should be performed before the growth has become very extensive, and the defect filled in by a plastic operation. After an apparent cure by any means the patient should be watched for at least a year because of the danger of recurrence.]

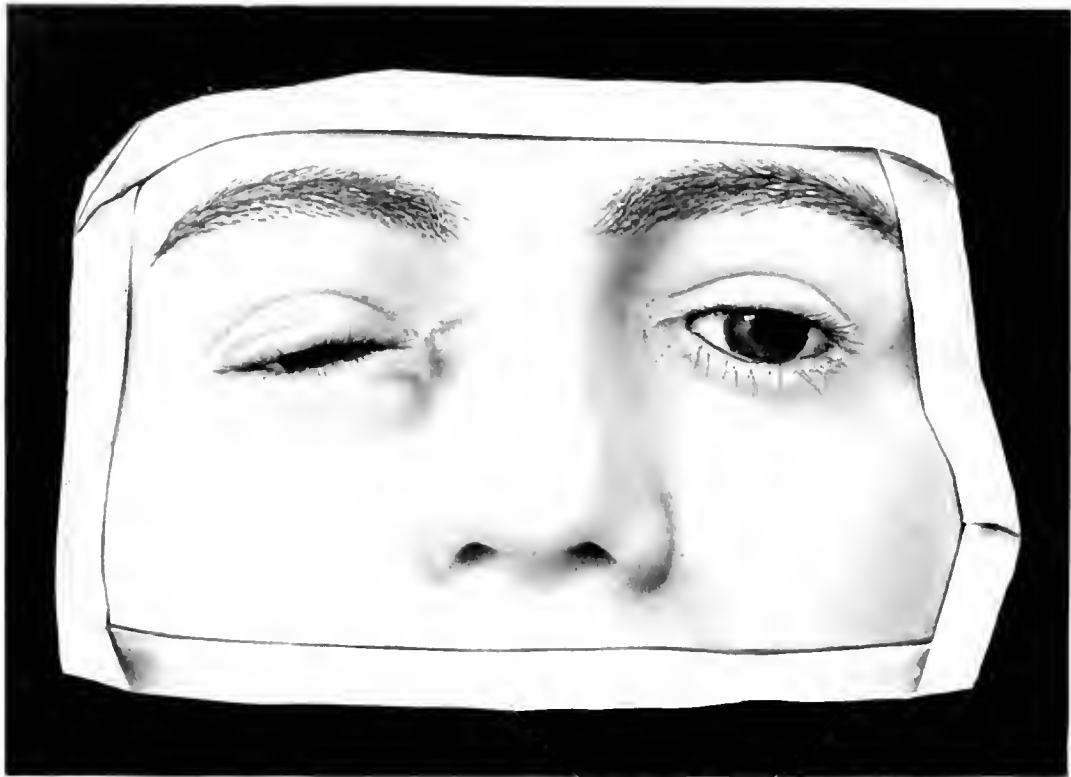


Fig. 22. Dacryo - Cystitis acuta

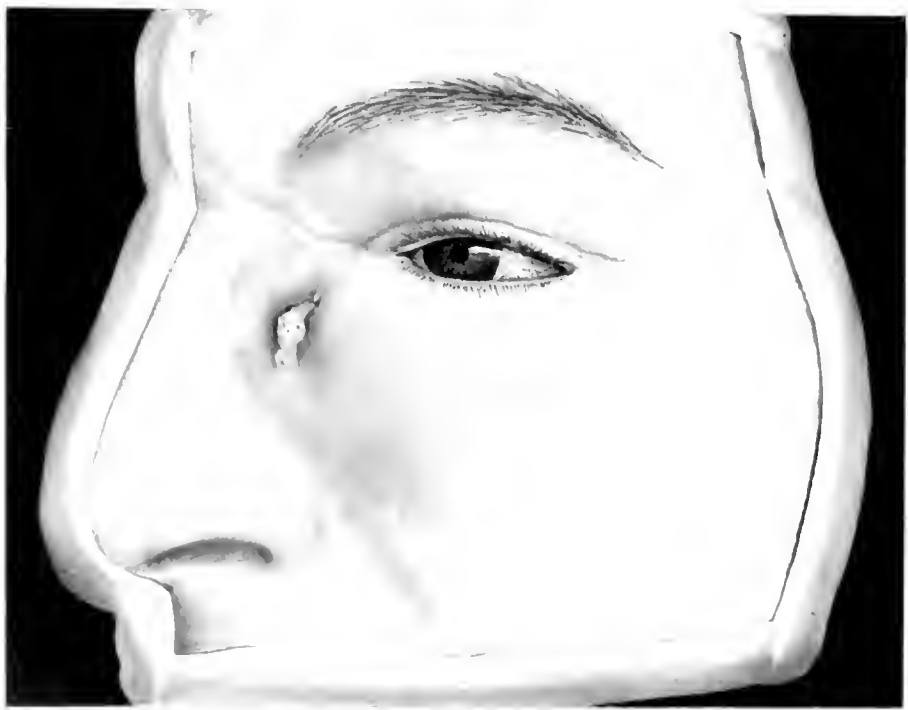


Fig. 23.
Dacryo-Cystitis with Fistula.

Dacryocystitis.

Plate XVIII, FIGS. 22 AND 23

Dacryocystitis is an inflammation of the lachrymal sac. Such an inflammation is very rarely due to an extension downward of an inflammation of the conjunctiva, but is produced almost always by a transmission upward of some disease in the nose, by the decomposition of lachrymal fluid accumulated above a stricture in the duct, or by disease of the neighboring bones.

Cause

In the majority of cases the cause is a stricture of the duct, above which the lachrymal fluid stagnates. This condition may last a long time with no externally visible signs of inflammation, except that the eye is frequently bathed in pus that has been secreted by the mucous membrane of the sac and has passed back through the canaliculi. This pus may be made to regurgitate at almost any time by pressing the finger upon the lachrymal sac, when a muco-purulent secretion will be seen to exude immediately from the punctum. This disease is called **chronic dacryocystitis**.

The **virulence of this muco-purulent secretion from the lachrymal sac** was well known long before the days of bacteriology. As long as an eyeball is intact it may be bathed with this pus with impunity, but if a trivial wound of the epithelium allows access to be gained to the tissue of the cornea a white spot appears on its surface and develops into a serpiginous ulcer. The reason of this is now known to be that the pus in the lachrymal sac usually contains very virulent pneumococci, sometimes in pure cultures.

A similar inflammation is produced in **tuberculosis** when a tubercle or a tuberculous ulcer is formed in the wall of the sac, and also in old **trachoma**, when the sac wall is found to contain trachoma granules.

Treatment

The nasal cavity should be investigated and any pathological condition therein which might give rise to, or maintain a dacryocystitis

should be remedied, [but this rarely suffices for a cure. Local treatment is imperative in almost all cases; this may be conservative or radical. By **conservative treatment** is meant any form in which the attempt is made to preserve the lachrymal passages and to maintain the flow of tears through them; by radical, any form in which the natural conduit of the tears is abandoned.

For the purpose of examination the pus may be pressed out of the sac, the canaliculus dilated with a conical probe, and the attempt then made to wash out the passage with some weak antiseptic solution, but such an attempt will succeed only in very exceptional cases, because a stricture is usually present. It is not wise to try to pass a probe through a dilated canaliculus down into the duct, because the mucous membrane is very likely to be injured and the condition to be made worse, although in very skillful hands it may sometimes be done for diagnostic purposes. It is much better to slit the canaliculus and to incise the stricture before the probe is passed. This is commonly known as *Bowman's* operation.

The lower canaliculus is the one usually operated on. The skin of the lower lid is drawn outward so as to put it on the stretch and to bring the punctum into plain view; the probe point of a *Weber* knife is introduced vertically into the punctum, the knife is then brought to a horizontal position with its edge directed toward the inner lip of the margin of the lid, pushed forward along the canaliculus until its point impinges firmly on bone, and then, while contact with bone is maintained, the handle is raised to a vertical position so as to slit the entire canaliculus. The knife is then rotated until its edge is directed forward, pushed down through the lachrymal sac and duct, cutting the stricture, and withdrawn. From this point on all manner of modifications have been made as regards the use of small and of large probes, the practice of intermittent probing, the introduction of styles and canulae to be worn constantly, and the irrigation of the canal with various drugs, but none of them have given perfect satisfaction. I prefer to pass as large a probe as can be inserted without undue violence at the time of the operation, and afterward to pass a slightly smaller one at first daily or every other day, then at longer intervals, leaving it in for 5 or 10 minutes. Others prefer to begin with a small probe and to gradually increase the size. It does not seem to make much difference what solutions are used for irrigation, provided they are not of such a nature, or so strong as to do damage. *Roemer* prefers a solution of pneumococcus serum, if pneumococci are present, but has not found it very satis-

factory. In spite of all the skill and care that can be exercised the after treatment is usually prolonged for months, the probing is very painful, unless we take the very decided risk of creating a cocaine habit by applying that drug to the lachrymal passages at each visit, and the disease is not cured in a large percentage of the cases.

Very recently *Wessely* has recommended the injection into the sac of a few drops of tincture of iodine. He says that the immediate results were excellent in 80% of his cases, but sufficient time has not elapsed for us to know anything of the final results.

Operative treatment from within the nose has also been tried and performed by *West* in over a hundred cases with many excellent results. The great advantage of this method of treatment is that the function of the lachrymal passages is preserved. It is indicated in all forms of stenosis of the duct, but contraindicated in very young children, and in very old people.

Radical treatment consists of extirpation of the lachrymal sac, or of the formation of an artificial outlet for the tears into the nose. Both give better and quicker results than *Bowman's* operation, and cause the patient much less pain.

For **extirpation** an incision is made over the lachrymal sac, beginning about 0.5 cm. above the internal palpebral ligament, curving gently downward and outward 2.5 cm. to a point a little above the commencement of the bony canal. The muscular tissues are separated down to the fascia covering the sac, which is exposed by retracting the edges of the wound, and traced to the anterior lachrymal crest, from which it is torn away. The sac is then separated from the structures on its nasal side by blunt dissection upward to the internal palpebral ligament, which crosses its upper part. With some trouble the apex of the sac may be dissected out from beneath this ligament, but the division of the ligament seems to do no harm and facilitates the work. The sac is then seized with forceps, its close attachments to the tissues on its outer side are divided with a knife or with scissors, great care being taken not to wound it, and, when free elsewhere, is cut off as closely as possible to the bony canal. The mucous membrane of the duct is then curetted away thoroughly with a sharp spoon, the wound is washed out with saline, its edges sutured together, a pad of aseptic gauze accurately adjusted over the region of the sac, and a bandage applied firmly enough to maintain pressure. This dressing may be removed with the sutures in from 5 to 7 days, but it is well to apply collodion for a few days longer.

The principal, if not the only objection to this operation, for the resultant scar soon becomes almost imperceptible, is that while the outlet for the tears from the eye has been destroyed, nothing has been provided in its place. Unless the palpebral lachrymal gland is removed at the same time epiphora must result and the patients be annoyed more or less by the overflow of tears down the cheek. As epiphora is sometimes the symptom that leads the patient to the surgeon, it may happen that its most annoying symptom may persist after the disease has been cured. Hence many attempts have been made to create a new outlet for the tears into the nose, but they were unsuccessful until *Toti* established a permanent drain by the operation known as **dacryorhinostomy**.

An elliptical incision 3 to 3½ cm. long is made through the skin, soft tissues, and periosteum about the inner canthus, passing 3 mm. in front of the lid attachment of the internal palpebral ligament and enclosing the entire lachrymal apparatus. The flap thus marked out is elevated with the periosteum laying bare the bone as far as the posterior lachrymal crest. When done under local anaesthesia a tampon of adrenalin and cocaine should now be placed in the cavity thus formed, while adrenalin and cocaine are applied in the nose. *Török* thus describes the resection of the bone:

“The edges of the bone to be resected correspond entirely to the detachment of the periosteum, that is: We resect first the edge of the orbit at the extension of the fossa sacci lachrymalis, then we resect an oval piece of the bone which is bordered by the crista lachrymalis posterior posteriorly, the ductus naso-lachrymalis inferiorly, and the elongation of the crista lachrymalis posterior superiorly. Care must be taken not to injure the nasal mucous membrane. Exceptionally we may find some ethmoidal cells behind the anterior segment of the piece of bone to be resected; we may encounter behind the superior segment some of the inferior frontal cells, or behind the posterior segment some of the lachrymal cells. They may be resected.”

The entire inner wall of the sac is then removed and an aperture made in the nasal mucous membrane exposed by the resection of the bone which will correspond exactly with the opening in the lachrymal sac. Before making this aperture it is well to push the nasal mucous membrane through the hole in the bone, at the same time bringing the remaining wall of the sac toward it in order to see that they come together easily; if they do not it may be necessary to enlarge the opening in the bone. The edges of bone are then smoothed off, the

incision in the skin closed with sutures that take in as much as possible of the underlying tissues, a gauze tampon is placed in the nose to press the two membranes together, and a sterile gauze dressing is bandaged on. It is often necessary to remove the middle turbinate.

The patient must remain quiet and not blow his nose. If no complication ensues the tampon may be removed on the fourth day, the dressing and sutures on the eighth.

The number of cases reported in which this operation has been performed is not very great, but the results seem to have been perfect in from 50 to 60%. Very few total failures are recorded, but epiphora persists in many. Bacteria disappear more slowly than after extirpation of the sac, so the latter is preferable when an operation on the eyeball is contemplated.]

CONGENITAL DACRYOCYSTITIS

Pus is not infrequently seen to come from the lachrymal sac of an infant a few days after birth, when there is danger of an erroneous diagnosis of ophthalmia neonatorum, unless a careful examination be made. Sometimes the children are suffering from congenital syphilis, when the suppuration may be the result of disease of the neighboring bones and can often be cured by antisiphilitic treatment alone. In other cases the condition is one of simple retention due to an occlusion at the lower end of the duct, and is not one of blennorrhoea. Often an energetic pressure upon the dilated sac suffices to open the channel and to cause the pus to disappear. [When this procedure is not successful at first the mother may be taught to press out the pus from the sac regularly, for the duct opens spontaneously in the great majority of cases. If the occlusion should persist obstinately the canaliculus may be dilated and a probe passed, but this should be done with the greatest care, and only when further delay seems hopeless, because of the danger of inflicting serious damage upon the mucous membrane.]

ACUTE DACRYOCYSTITIS

The picture is quite different when from any cause the pus breaks through the wall of the sac and spreads rapidly beneath the loose adjacent tissues. The lids become oedematous, and soon we have a subcutaneous abscess with the dilated lachrymal sac in its center. This condition is known as **acute dacryocystitis**, although it is

anatomically a pericystitis, or a cystitis with rupture, and is exemplified on Plate XVIII, Fig. 22.

Diagnosis

At the site of the lachrymal sac may be felt an induration which is so exquisitely tender that the patient often draws back in pain as soon as it is touched. Both lids and the cheek may be greatly swollen, and in many cases a very noticeable feature in the region of the greatest swelling is a deep, horizontal furrow separating two tense, red protuberances (see Fig. 22). This furrow is formed by the internal palpebral ligament, which arises from the inner canthus, passes over the lachrymal sac, and is inserted into the lachrymal bone. [In acute dacryocystitis the greater of these two swellings is below the ligament, while in a ruptured empyema of the ethmoid or frontal sinus the greater swelling is above.]

Treatment

The treatment should be that of an abscess. No attempt should be made to wash out or to probe the lachrymal passages during the inflammatory stage lest the infection be carried elsewhere.

Sometimes the abscess may be induced to undergo resolution by rest in bed and hot, moist applications, but if this does not happen, the pus finally breaks through the skin and escapes (see Plate XVIII, Fig. 23). It is better to anticipate this by a free incision, after which healing commonly follows. When suppuration persists it is usually because of caries of the lachrymal bone, and in these cases it is needful later to reopen the wound widely, curette the bone and pack with ganze, but a fistula of the lachrymal sac may remain after all of these operations. [To cure a fistula it is necessary to extirpate it together with the lachrymal sac.]

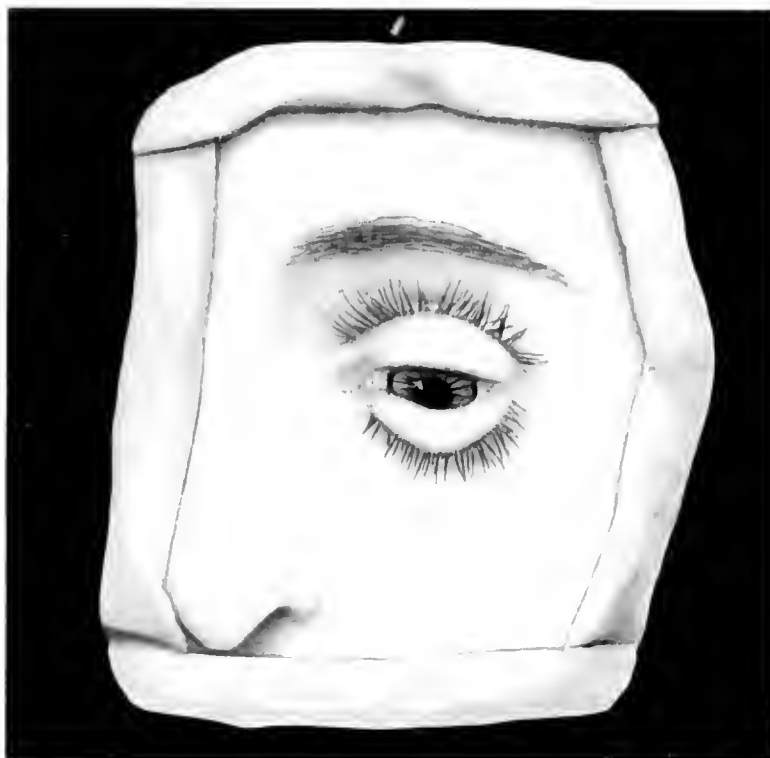


Fig. 24.
Conjunctiva. Normal Condition.

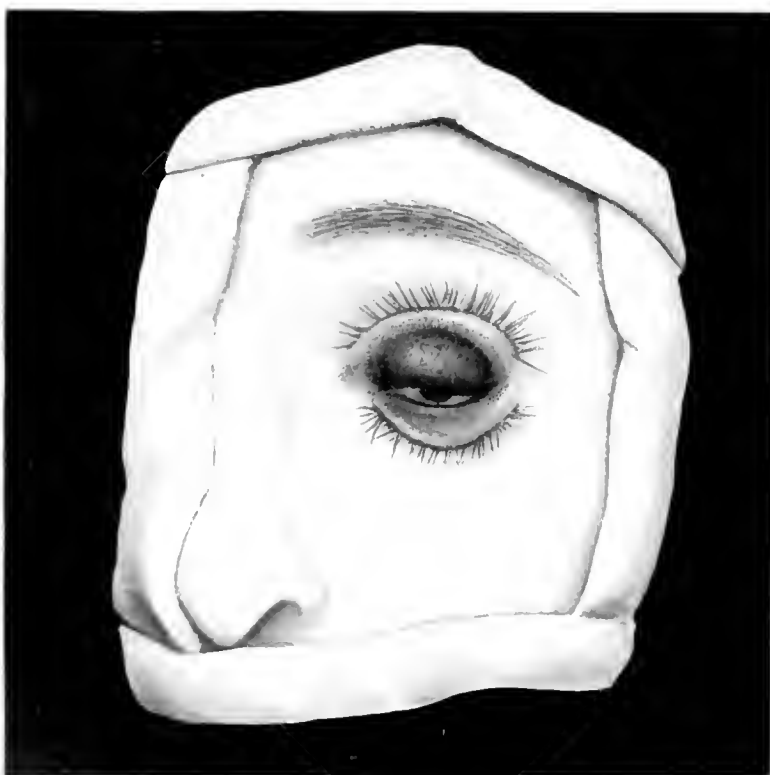


Fig. 25. Conjunctivitis catarrhalis simplex.

Normal Palpebral Conjunctiva.

Plate XIX, FIG. 24

The conjunctiva forms a sac with an aperture corresponding to the palpebral fissure, and blends with the skin at the margins of the lids. It is divided into three portions: 1, the palpebral conjunctiva; 2, the transition fold, or the conjunctiva of the fornix; 3, the bulbar, or scleral conjunctiva.

The **palpebral conjunctiva** has the characteristics of a true mucous membrane, and we accordingly find it alone the seat of follicular diseases, and of the infectious diseases peculiar to mucous membranes, such as gonorrhoea and diphtheria. The **bulbar conjunctiva** on the other hand resembles epidermis, and therefore cutaneous diseases, like eczema, attack the eyeball directly.

The palpebral conjunctiva, as it appears on the everted lids, is shown in Fig. 24, Plate XIX. The mucous membrane is pale and smooth, and allows the yellowish Meibomian glands beneath it to be distinctly seen.

Catarrhal Conjunctivitis.

Plate XIX, FIG. 25

The palpebral conjunctiva is so delicate and sensitive that a slight cause, such as contact with the air, or with water, eversion of the lids, or weeping, excites in it hyperaemia and a condition of distinct irritation. A prolonged irritation of this kind, or one more intense in its nature, results in an inflammation, which takes the form of a chronic, or of an acute catarrh. The surface of the swollen and inflamed conjunctiva is then no longer smooth, but is covered with minute elevations and depressions that give it a finely granulated appearance, or one that resembles cut velvet (see Plate XIX, Fig. 25). When the elevations are large they are called papillary, and are to be carefully differentiated from the follicles described under follicular conjunctivitis.

Modern bacteriology has taught us that an **acute conjunctivitis** is almost always due to infection with some such microorganism as the pneumococcus, the Morax-Axenfeld bacillus, the Koch-Weeks bacillus, or the streptococcus, each of which produces a clinical picture that differs somewhat from those produced by the others. Acute conjunctivitis is contagious, and sometimes appears in the form of a widespread epidemic of acute disease; in this respect it contrasts strongly with trachoma, for which it has been frequently mistaken.

Chronic conjunctivitis is caused by such external irritants as dust, smoke, wind, and cold, or it may follow an acute infection.

Treatment

The indications in **acute conjunctivitis** are to disinfect the conjunctiva, to remove the secretion, and to do away with the dilatation of the blood vessels. *Nothing is more senseless than to bind up the eye when thus inflamed.* If the secretion is allowed to stagnate the very best of media is furnished for the rapid multiplication of the microorganisms. It should be allowed to escape freely, and should be washed out at frequent intervals with sterile water, or some mildly antiseptic solution, like one of boric acid, or of sublimate, 1 to

5000. The fluid used for irrigation should be cold, so that the astringent effect of cold may be utilized. [This irrigation should be performed several times a day, often enough to keep the eye fairly clean. Its effect is purely the mechanical removal of the infectious secretion; the mucous membrane cannot be sterilized by any solution which will not injure the eye. After the irrigation the skin of the lids should be wiped dry and a little vaseline applied to prevent excoriation. A little vaseline on the edges of the lids at night tends to keep them from becoming stuck together. Many cases recover under this simple treatment alone.

A bacteriological examination should be made of the secretion in every case, not only to exclude the presence of gonococci, or other agents that excite a more serious inflammation, but also to detect the presence of microorganisms that demand a special treatment.

Ice cloths are of benefit during the acute stage of inflammation, although thermometric observations have shown that cold thus applied does not affect the temperature of the conjunctiva, but ice should never be applied, as there is some danger of freezing the skin of the lid and of causing gangrene. If a piece of cloth, or a pledget of wet cotton, or gauze, is allowed to freeze to the surface of a piece of ice, then torn off and laid on the eye for a few seconds, to be replaced by another, and this is kept up steadily for from 15 to 30 minutes, the patient will be made more comfortable at least, whether the acute stage is shortened or not. They should be discontinued as soon as the swelling of the lids and the acute symptoms abate.]

The sovereign remedy when the secretion is purulent is a $\frac{1}{2}$ to 1% solution of **silver nitrate** painted on the conjunctiva of the everted lid. [The stronger solutions should be neutralized with salt and water after application before the lid is allowed to return to its normal position.] Various substitutes for silver nitrate have been advocated, such as protargol, argentamin, albargin, [and argyrol. They are fairly efficient and are usually preferable in this class of cases because they are far less irritating and can be prescribed for the patient to have a drop instilled into the conjunctival sac two or more times a day. When the inflammation is due to the Morax-Axenfeld bacillus zinc sulphate is our main reliance. The eye may be cleansed with a weak solution of this drug, a drop of a stronger solution, from $\frac{1}{4}$ to 2%, instilled at regular intervals during the day, and an ointment, about $\frac{1}{4}$ %, applied to the lids at night.

After the secretion has nearly or quite ceased we should use some astringent for a while to contract the blood vessels and if possible

prevent the sequel of a chronic conjunctivitis. For this purpose a drop of any of the following solutions may be instilled once a day or oftener.] Zinc sulphate, $\frac{1}{4}$ to 2%; zinc sozoiodolate, $\frac{1}{2}$ to 1%; alum, 1%; sodium sozoiodolate, 3 to 6%; resorcin, 1 to 2%; tannic acid, 1 to 2%.

[**Chronic conjunctivitis** is usually treated with such astringents as those named above. Many of the cases are extremely obstinate; often it is an occupational disease and cannot be cured unless the patient changes his mode of life. Trouble in the lachrymal sac, in the margin of the lid, and in the ducts of the Meibomian glands must be sought for and treated if found. Finally the *refraction* should be carefully corrected, for an uncorrected error is the cause in a certain number of cases.]

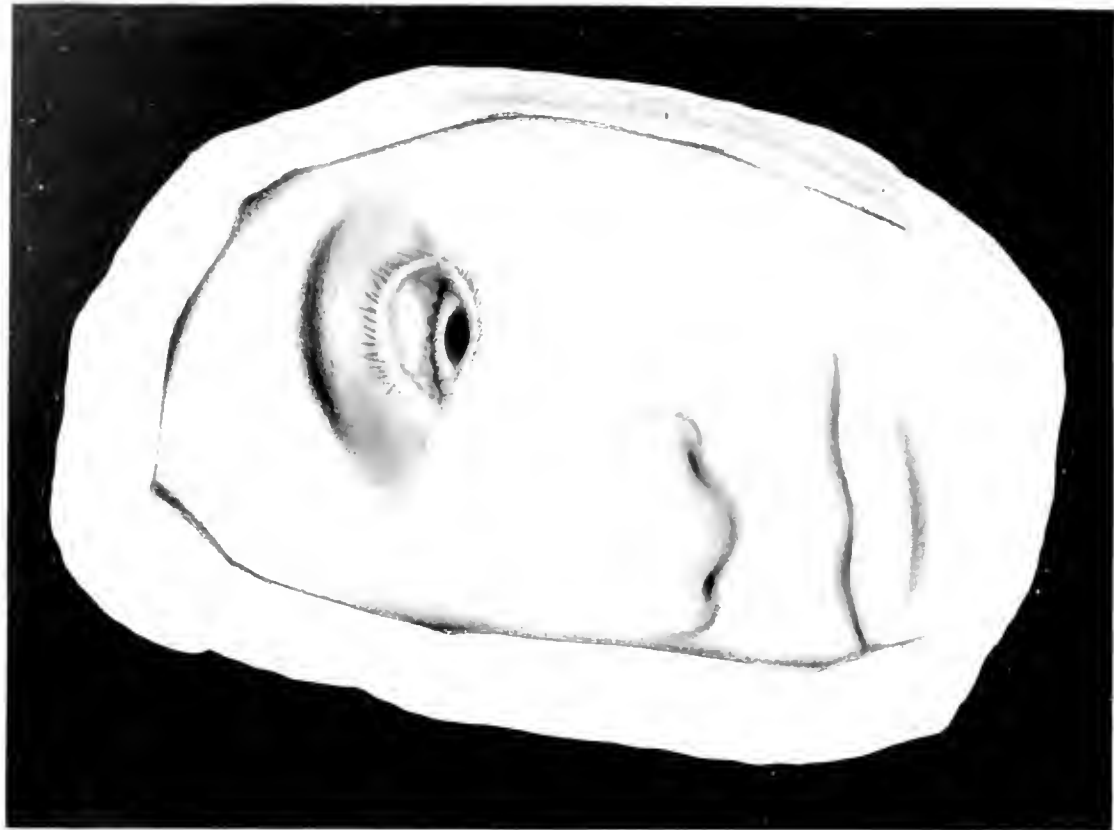


Fig. 27.
Conjunctivitis trachomatosa I.

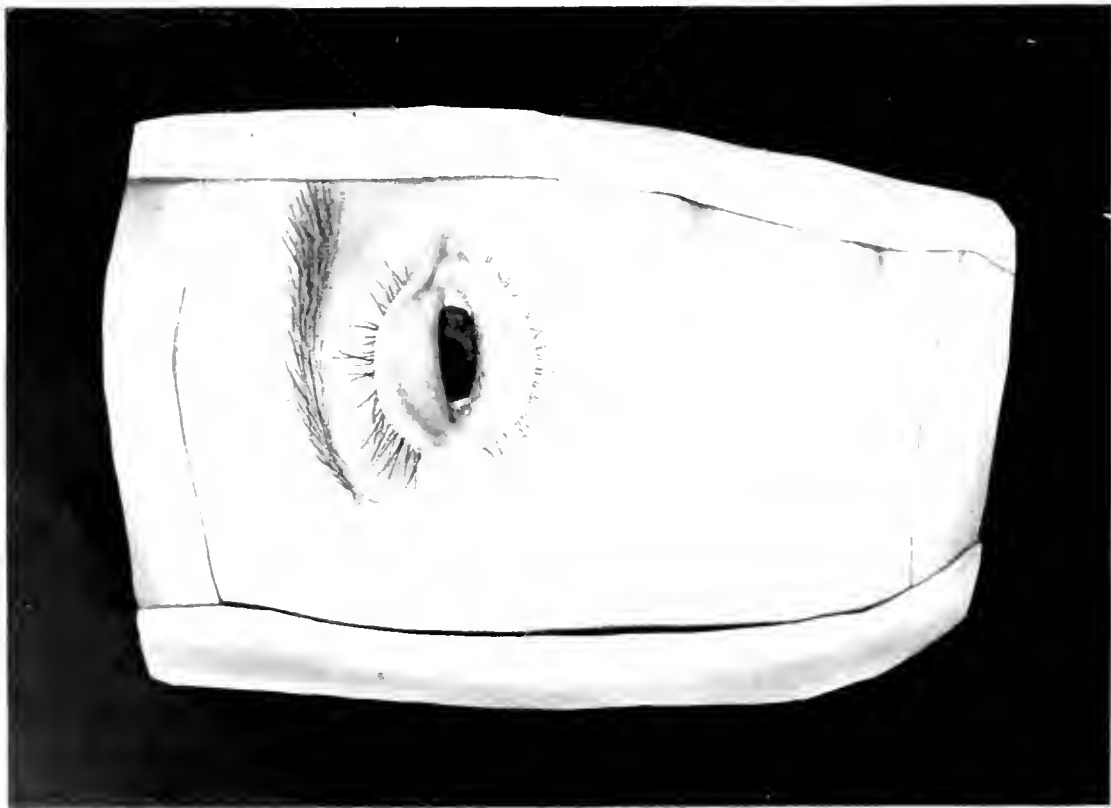


Fig. 26.
Conjunctivitis follicularis

Follicular Conjunctivitis.

Plate XX, FIG. 26

Follicles are conjunctival elevations composed of circumscribed masses of round cells beneath the epithelium, and therefore are neoplasms similar to lymph follicles, or lymphomata. At first they are clear and vesicular in appearance, but later, particularly in the malignant forms, they resemble frog spawn, or grains of cooked sago. They are caused to develop by certain, but not by all, chemical, thermic and bacteriological stimuli, and their development seems to depend on the specificity of the stimulus, rather than on its intensity or duration. They are not produced by gonorrhoeal and diphtheritic infections of the conjunctiva, which are the most violent with which we are acquainted, nor by a single conjunctival catarrh, no matter how long it may last, and yet they occur in quite a number of conjunctival affections of both infective and non-infective origin, so that the varieties of conjunctivitis may be divided into those that are, and those that are not associated with the formation of follicles.

A certain percentage of the children in every school will be found to have follicles in the conjunctiva, although this membrane is otherwise normal. In these cases a diagnosis of follicular conjunctivitis is wrong because there is no conjunctivitis. These follicles give rise to no trouble. They may be simply dilated superficial lymph vessels, lymphectasiae, when the children are chlorotic or anaemic. Follicles develop wherever many persons live crowded together in impure, stagnant air, as in overcrowded school rooms, orphan asylums, and prisons, and may be found post mortem with the microscope in the conjunctivae of all prisoners who have been confined for a long time. As there is usually no sign of inflammation in these cases I believe that the condition is due not to infection, but to a reaction from the irritation produced by the air. Some authors ascribe the irritation to the ammonia in the air, others to an anthropotoxin.

Follicles also occur in certain forms of conjunctivitis of infectious origin, but as a rule they are not very numerous, are superficial, and are to be found mainly in the conjunctiva of the lower lid. They disappear spontaneously, leaving no scars.

[They are sometimes excited also by the prolonged application of certain drugs to the conjunctiva, such as atropine, eserine, and the red oxide of mercury. These drugs may have been used for weeks, and may have seemed to be well borne, when a conjunctivitis sets in for which no bacterial cause can be detected, associated with rows of follicles in the lower transition fold. These follicles disappear completely soon after the use of the drug has been discontinued.]

Treatment

Open air and exercise are often sufficient to cure school folliculosis. The follicles so common among school children frequently disappear during vacation, but it is well to wash the eyes at intervals with a 2 to 4% solution of boric acid, or with lead water. Compresses wet with the same may also be applied. Perhaps the best collyrium is the 2% solution of borax recommended by *Foerster*.

[When a conjunctivitis is present the alum pencil may be applied two or three times a week, or one of the astringent solutions mentioned under the treatment of catarrhal conjunctivitis may be employed.]

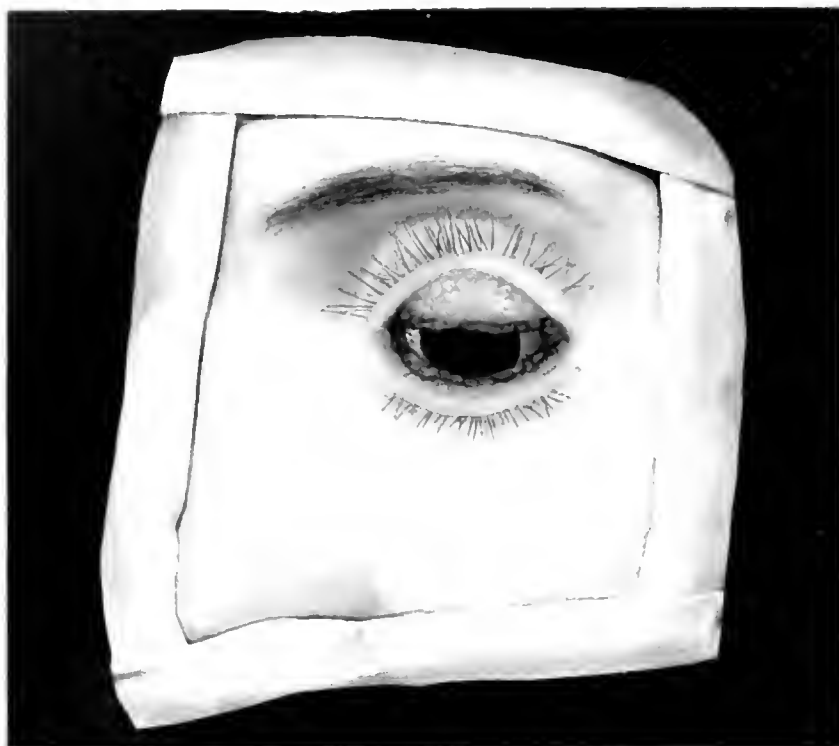


Fig. 28. Conjunctivitis trachomatosa

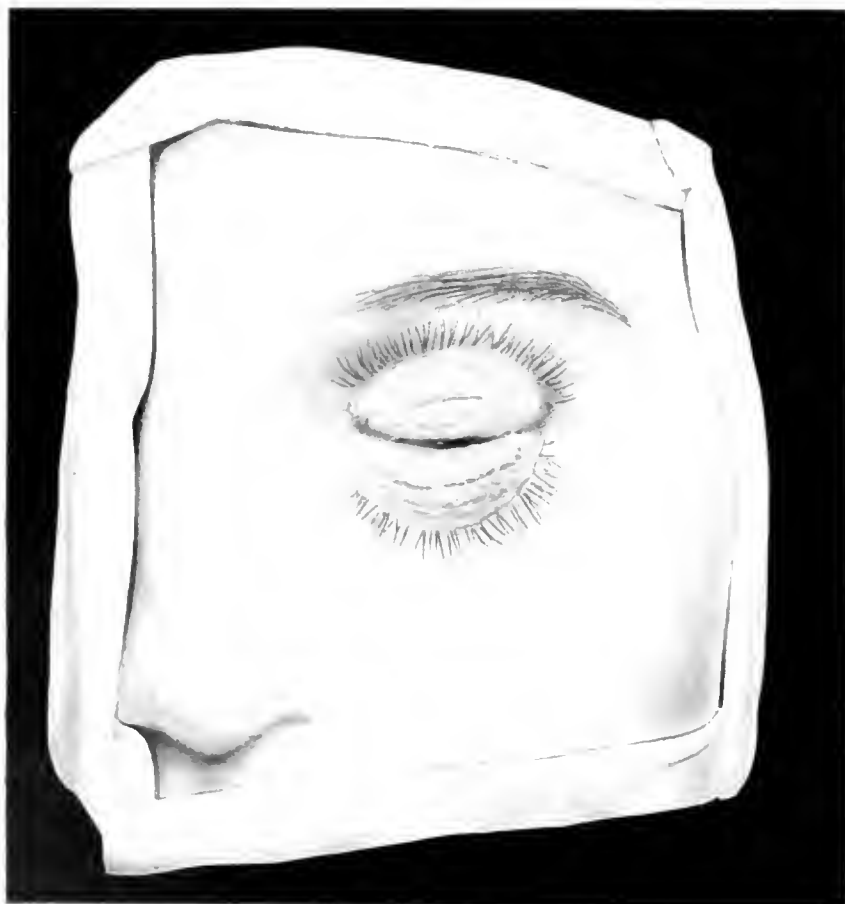


Fig. 29. Sklerosing Trachoma.

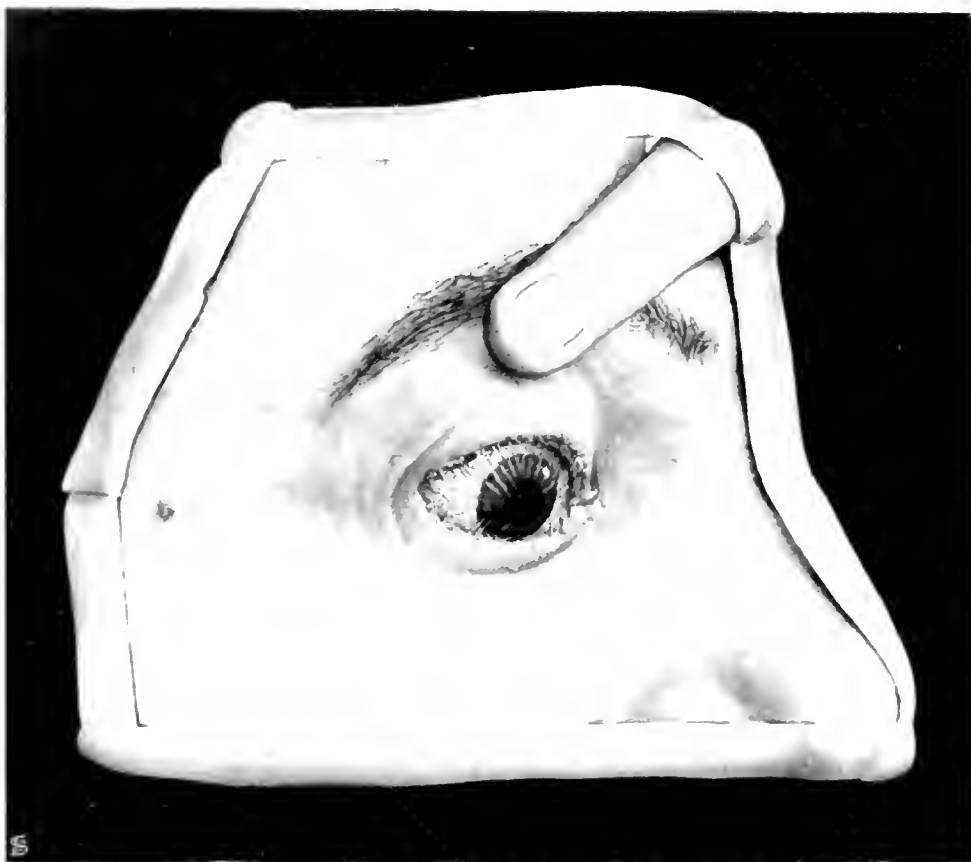


Fig. 30. Pannus trachomatosis.



Fig. 31. Entropium and Trichiasis through scar-tissue.



Fig. 33.
Xerophthalmus trachomatosis

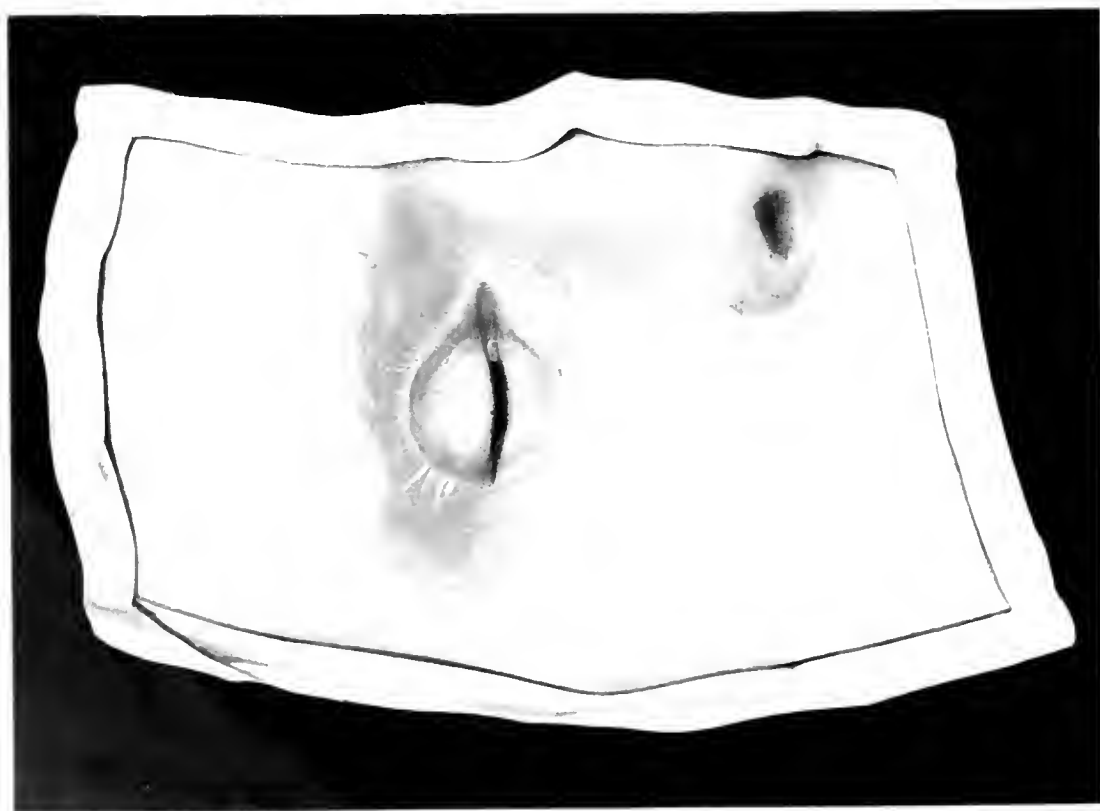


Fig. 32.
Trachoma cicatricum

Trachoma.

Plate XX, FIG. 27; *Plate XXI*, FIGS. 28, 29; *Plate XXII*, FIGS. 30, 31; *Plate XXIII*, FIGS. 32, 33

Trachoma is a chronic, infectious disease of the palpebral conjunctiva peculiar to man and not communicable to the lower animals, except perhaps in a very mild form to the anthropoid apes. It is spread over the whole world, but is not uniform in its distribution. In some places it is absent, in others it is endemic, while within regions free from the disease are to be found islets of varying size in which it abounds. Wherever trachoma is endemic it is progressive, slowly infecting one person after another, as it has done perhaps for centuries. It is only in rare cases that the onset may be acute. The disease is contagious and the means of contagion most to be feared are dirty washbasins and towels, but infection does not take place readily; it usually occurs only after continuous long contact with trachomatous persons, or the use in common with them of beds and washbasins. A long time may elapse after infection has taken place before the patient notices the least trouble with his eyes, and it may be months or years before the process becomes malignant. In a family of 6, 3 perhaps may be affected and it may be months or years before the disease appears in the others. If an individual suffering from trachoma is shut up with other persons in an institution, some of his associates will become infected in the course of time, and a little endemic will gradually develop. I have never yet found all the inmates of such an institution affected, which goes to show that infection does not necessarily follow prolonged daily contact. The disease is always present wherever it is endemic, slowly passing from one person to another, while others recover, so that the number of sufferers varies from time to time within certain limits; in some years the number is greater, in others less, but reports that trachoma has broken out suddenly in any place have always been proved incorrect. Either attention had suddenly been drawn to it where it had existed for years, or the disease was not trachoma.

The **specific agent** is not yet known with certainty. [It was thought for a time that this agent had been found in the minute

bodies, smaller than any known cocci, discovered by *Halberstaedter* and *Prowazek* in the cells of the follicle contents. These bodies, or cell inclusions, lie near the nucleus and multiply until they fill the cell, cause it to rupture, and are set free in the secretion. They are to be found chiefly in cases of fresh, untreated trachoma, less easily when the disease is of long standing, but thus far all attempts to cultivate them have failed. Similar, if not identical bodies have been found in the non-trachomatous conjunctiva and in other mucous membranes. At present it is uncertain whether they are microorganisms, or products of degeneration.]

The disease is characterized by large follicular **granules**, which grow larger and more numerous, with signs of inflammation, great papillary swellings, to finally necrose and cause subepithelial contractions. It is not always possible to differentiate it at once from follicular conjunctivitis, but the diagnosis can always be made after prolonged observation. In follicular conjunctivitis the granules are absolutely benign, cause no inflammatory reaction, or swelling of the palpebral conjunctiva, and no great swelling of the transition folds and papillae, but are apt to be seen in pale, anaemic membranes. Sooner or later they disappear, having done no damage and leaving no traces. While they are clear, pale, and resemble vesicles, the more deeply seated trachoma granule soon presents a gray, discolored appearance, and, in well marked cases, an inflammatory reaction in its immediate vicinity. It seems to become sunken in the adjacent red and swollen conjunctiva, while the enlarged, thickened transition folds protrude when the lids are everted. Large papillary proliferations soon appear as evidence of the reaction, which is never present in follicular conjunctivitis. Finally the granule proves its malignancy by destroying the conjunctiva in which it lies, transforming it into a very dense connective tissue. It produces a sort of cirrhosis, similar to that caused by certain inflammations in the lungs, liver, and kidneys, which is responsible for most of the sequelae of trachoma. The cornea is frequently involved with the production of pannus.

Course

The course of trachoma is likewise insidious and chronic, and may last a lifetime if untreated, yet quite a number of patients finally recover.

After the granule has existed a long time a softening takes place in its interior; the large cells necrose, their nuclei no longer staining,

and finally a granular mass is formed in which the constituents can no longer be made out. In many cases this softening does not involve uniformly the entire contents of the follicle, but begins in foci. As a rule it is most marked in the center. The most typical example of the softened granule is to be seen in the so-called gelatinous trachoma of *Stellwag*, in which the individual, softened granules are packed closely together and seem to blend (see Fig. 29).

According to *Raehlmann*, *Addario*, and others, the natural **termination** of the process is the rupture of the granule and the escape of its contents, but it seems to me that the gradual absorption of the contents is by far the more common and natural ending. This absorption certainly takes place not only when the contents are softened, but at any stage, as is shown by clinical experience. In very many cases we have seen little follicles, in which softening of the contents was out of the question, gradually undergo involution under the influence of medicamental treatment, although no external rupture took place.

Trachoma is one of the most malignant diseases of the eye and, like ophthalmia neonatorum, contributes enormously to the numbers of the blind in the so-called trachoma regions, while the number is still greater of those who have to suffer in later life from its sequelae, without becoming actually blind. Even the best treatment must be very energetic and maintained for months, if a positive and permanent cure is to be obtained. Relapses are very common.

TRACHOMATOUS PANNUS

Trachoma may pass from the conjunctiva of the lids and fornix to the cornea, skipping that of the sclera, and produce a **pannus** (see Plate XXII, Fig. 30). This usually begins at the upper margin of the cornea with the formation of little elevations that are at first so minute that they can be seen only with the aid of a magnifying glass, but grow to the size of poppy seeds. These gray white, solid nodules, consisting of distinctly circumscribed masses of lymphoid cells beneath the epithelium, may be called follicles. They are often surrounded by a slight opacity like a halo, when situated in the clear cornea. Later these nodules blend to form a diffuse, cellular, soft tissue which pushes down beneath the epithelium over the transparent cornea. This neoplastic layer is not of uniform thickness, so its epithelial covering presents an uneven surface. When this layer has advanced about 1 mm. a vascular proliferation starts from above to accompany it, but always remains a little behind the zone of infiltra-

tion. These vessels are not all directed meridionally toward the center of the cornea, but rather run in parallel courses from above downward.

The appearance of pannus varies with the vascular development. When it is fresh and the vessels are few it is known as ***pannus tenuis***; when the vessels are very abundant, as ***pannus vasculosus***; when the proliferation is so thick and vascular that the cornea seems to be covered with wound granulations, or raw flesh, as ***pannus crassus***, or ***carnosus***; when the pannus is old, with connective tissue transformation, it usually contains only a few slender vessels and is called ***pannus siccus***. The name pannus sarcomatosus, sometimes applied to pannus crassus or carnosus, is to be deprecated as misleading.

In the involution of pannus the zone of infection retires first and then the vessels, so that the latter are always a little behind the former and furnish a means by which it can be determined whether the pannus is advancing or receding. The picture changes as soon as the pannus has passed the center of the cornea. The vessels no longer come simply from above, but radiate from all directions over the surface. They often anastomose and form varicose swellings in the center of the cornea, and it can be perceived from their indistinct, bluish appearance that they lie more deeply in the tissue.

CICATRICIAL TRACHOMA

After it has existed for years, as a rule, trachoma finally causes a more or less extensive cicatricial contraction of the affected conjunctiva and later implicates the tarsus (see Fig. 32). The tarsus is at first densely infiltrated and swollen, later it shrinks and becomes transformed into a rigid, sclerotic, cicatricial tissue.

The **distortion of the tarsus** is typical in all cases. It not only exhibits a uniform deformation, but it becomes bent at an angle [about 3 mm. above the free margin of the lid]. The callous thickening of the conjunctiva is greatest at this bend and forms, as it were, the fixed point in the cicatricial contraction, toward which the two margins of the tarsus are drawn by the contracting force of the scar. Thus the trough-shaped, deformed tarsus is more or less bent at an angle at the densest place in the conjunctival cicatrix.

The free margin of the lid is bent inward along with the lower margin of the tarsus, producing the condition known as entropium (see Fig. 31).

Treatment

The treatment of trachoma may be divided into medicamental, mechanical, and operative. The two drugs most commonly employed to induce absorption of the granules are silver nitrate and copper sulphate. A 2% solution of the former is painted on the conjunctiva of the everted lids, especially when the secretion is abundant. The best way to use the latter is in the form of a crystal, the so-called **copper pencil**, or bluestone, which should be passed once a day gently and smoothly over the affected conjunctiva. Cold compresses may then be applied for a while and repeated after an interval if the irritation of the eye persists. If the physician does not see the patient every day he may prescribe a $\frac{1}{4}$ % solution of copper sulphate to be instilled twice a day, or a 1% ointment of the same. Many agree with *Arlt* in preferring copper citrate, usually in the form of an ointment, which can be obtained in tubes under the name of euprocitrol. Some of this may be rubbed daily into the conjunctiva with a glass rod. It is less irritating than the sulphate and therefore better fitted for home use.

Preparations of **iodine** have also been used a good deal. A collyrium may be made of 1 part of tincture of iodine in 15 parts of glycerine; iodoform may be dusted into the eye or applied in the form of an ointment; a $\frac{1}{2}$ to 1% solution of pure iodine in glycerine may be painted on the everted lids; or a caustic pencil may be made by moistening pure iodic acid with water until it forms a plastic mass that can be rolled on a glass plate, and applied to the conjunctiva every third day. The pain produced by the last is intense, but evanescent. [Comparing the results of treatment with iodic acid with those from other methods, *Rudas* claims that the length of treatment is shortened by about a third, that ulcer and pannus appear in 0.85% of the cases, instead of 3.25%, that disturbances of vision occur in 0.51%, instead of 3.07%, and that relapses take place less often, with a difference of 8%. The conjunctiva is cauterized and presents a raw surface after the reaction has passed off, so the same care must be taken for three or four days to prevent the formation of adhesions as after cauterization by other means. The after treatment is that for simple conjunctivitis, with the addition of euprocitrol. Cases suitable for this procedure are those with no inflammatory symptoms and no discharge.

Good results from the use of the **x-rays** have been reported by various writers. They have employed tubes of various spark gaps, and amperage of different strengths, at varying distances from the

eye, and given exposures of from 4 to 15 minutes. The lids are kept everted, the eye must be protected, and the adjoining skin should be covered with a lead mask. It is said to produce an infiltration of the conjunctiva that takes some weeks to subside, so treatment is stopped as soon as the granules have apparently been absorbed.

The efficiency of **radium** has been discredited by *May*, who obtained better results with the copper pencil.

Carbon dioxide snow applied for a few seconds to the cocaineized conjunctiva has recently been advocated. *Wibo* claims that two days after the application the granules have disappeared, leaving the conjunctiva smooth, but other advocates have found it necessary to repeat the application once a week for several weeks.]

Keinig's method is a good example of **mechanical treatment**. The diseased conjunctiva is rubbed hard with cotton wet with sublimate, 1 to 3000 or 1 to 5000, every other day until the granules have disappeared.

Operative measures shorten the treatment, which is very desirable when many patients have to be treated. Yet they are not all radical and it is wise to supplement them with medicamentous treatment. Fresh trachoma granules may be **expressed** with Knapp's roller forceps [or with the forceps devised for the purpose by *Noyes* and by *Prince*. Care must be taken that all of the granules are crushed and their contents evacuated. The conjunctiva is then washed off, some sterile vaseline applied, and the eyes bandaged for some hours.

The **galvanic cautery** may be used in two ways. Each granule may be punctured with a white hot platinum needle, but usually this has to be repeated a number of times, even when the cauterization seems to have been done thoroughly, and a cure cannot be obtained in this way when the tarsus is infiltrated. Good results have also been reported from the rapid passage of a broad, dark red cautery over the entire conjunctiva, but a considerable danger is incurred in this operation, for, if the cauterization should be too deep, a distortion of the tarsus with entropion would probably follow. Adhesions have to be guarded against. Healing takes place in 3 or 4 weeks.

In **grattage** the conjunctiva over the follicles is scarified with a knife and then brushed sharply with a stiff brush dipped in a solution of sublimate to remove the contents of the granules, or the contents may be removed with a small curette. Quite a number of instruments have been devised to incise or lacerate the conjunctiva and

evacuate the granules, but the results are not as good as could be desired, except in very mild cases.

In *Kuhnt's* expression a number of punctures are made in the infiltrated tarsus, one blade of a specially devised expressor is placed in the transition fold, the other on the conjunctival surface of the everted lid, and the two blades are then pressed together with considerable force, so as to cause the gelatinous masses to exude.]

When the infiltrations are old and deep the **excision operations** are indicated. [These are three in number, the simple excision, the combined excision, and the enucleation of the tarsus, are applicable to different stages of the disease, and are radical. The technique is difficult and needs the training of the specialist.

Simple excision may be performed when the disease exists mainly in the transition fold, and has not greatly implicated the tarsus. An incision is made from one canthus to the other in the healthy scleral conjunctiva close to the line demarking it from the diseased; the lower edge of the wound is slightly undermined and a few sutures introduced at the margin, to be passed later through the edge of the tarsus. Another incision separates the transition fold from the conjunctiva of the lid, and the ends of these two incisions are joined. The conjunctiva of the transition fold is then seized at the canthus with forceps and removed with scissors, sparing the subjacent tissue as much as possible. The sutures already in the scleral conjunctiva are passed through the margin of the tarsus and tied.

In the **combined excision** the transition fold and a part of the infiltrated tarsus are removed together. The first incision is the same as that for simple excision; three mattress sutures are introduced into the scleral conjunctiva on the lower side of the wound, so that each leaves a loop of thread on its outer surface, and, while traction is made on these sutures the scleral conjunctiva is dissected down to the limbus of the cornea. The second incision is made through the conjunctiva and tarsus about 2 mm. from the margin of the lid, the tarsus is seized with forceps, freed from all attachments on its outer surface and upper edge, and then the removal of the transition fold is completed. The patient is allowed to wink a few times, then the middle suture is drawn downward and forward, the lid seized with forceps exactly where the suture appears beneath it to indicate the spot where the needles shall be passed out through the skin of the lid just behind the cut edge of the tarsus. The ends of the suture are tied over a roll of cotton, and the other sutures in-

serted in a similar manner. After recovery the patient will have no trouble in winking if the flap of healthy conjunctiva dissected up from the eyeball is long enough to cover the cornea when allowed to hang down over it. The eye is bandaged for a couple of days, the sutures removed on the third. Medicamental treatment should be resumed at the end of a week.

Enucleation of the tarsus has been described under the treatment of cicatricial entropium. It is of good service when little is left of the transition fold and the tarsus is badly distorted.]

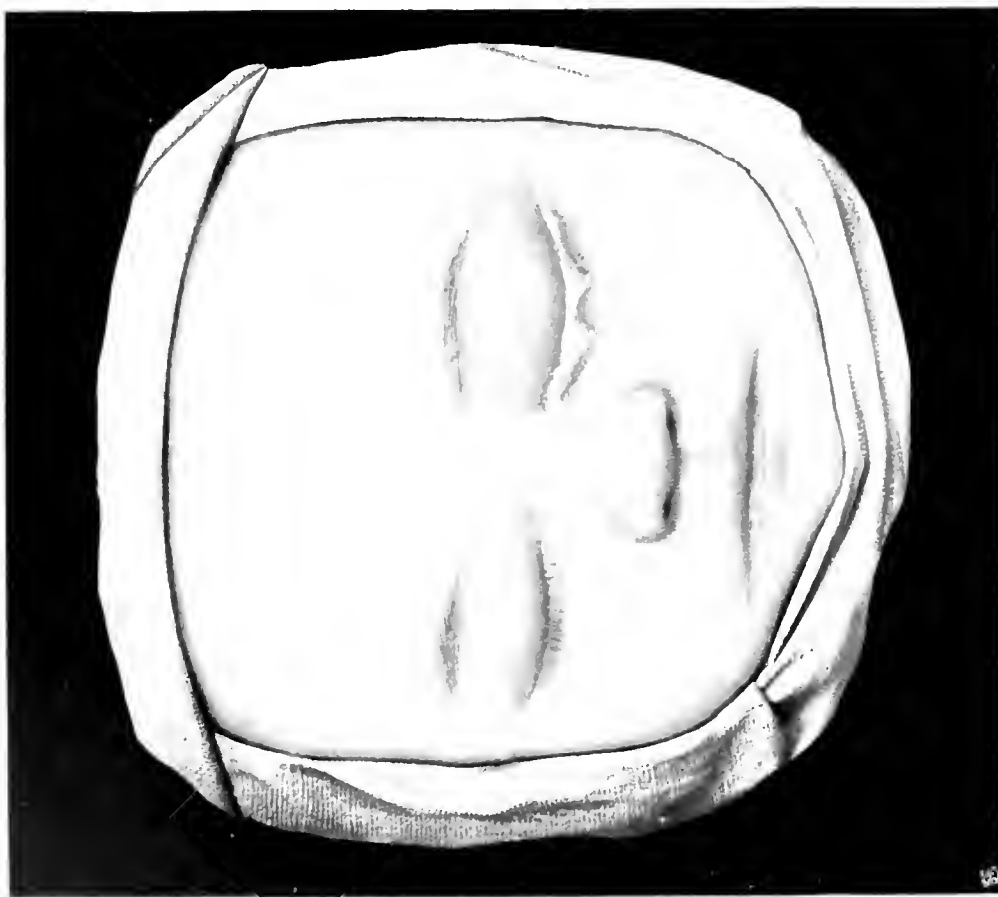


Fig. 34.
Conjunctivitis gonorrhoeica neonatorum
(Blephorrhoea neonat.)

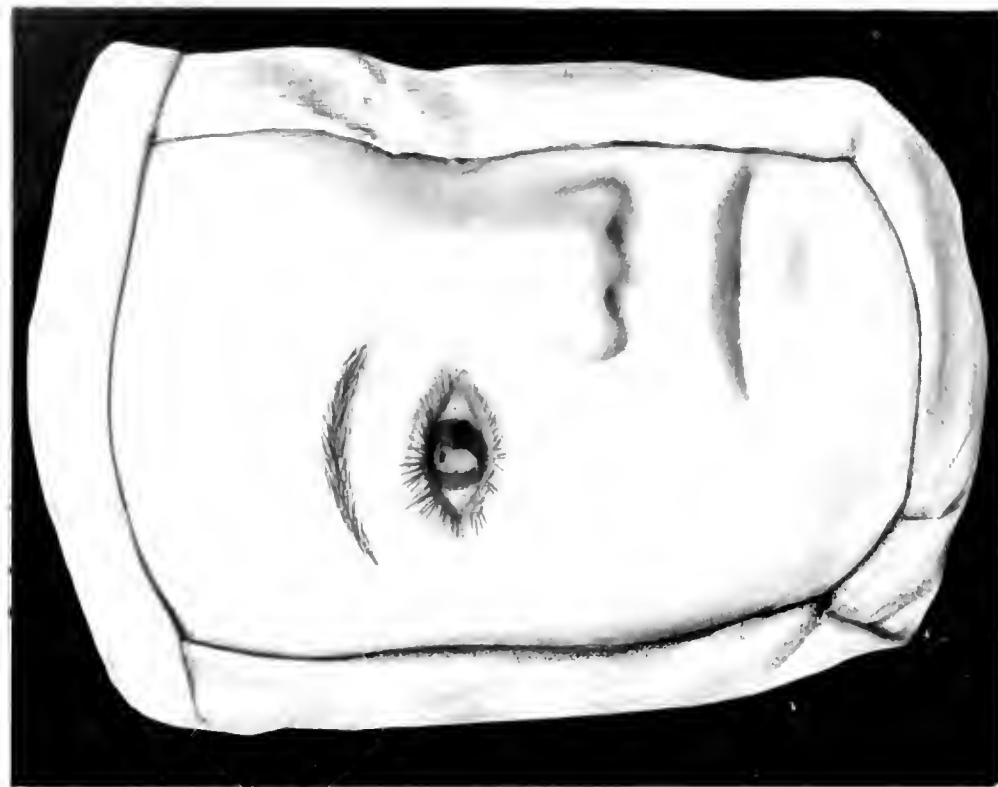


Fig. 35.
Leucoma corneae parziale. Coloboma iridis
artificiale following Conjunctivitis gonorrhoeica

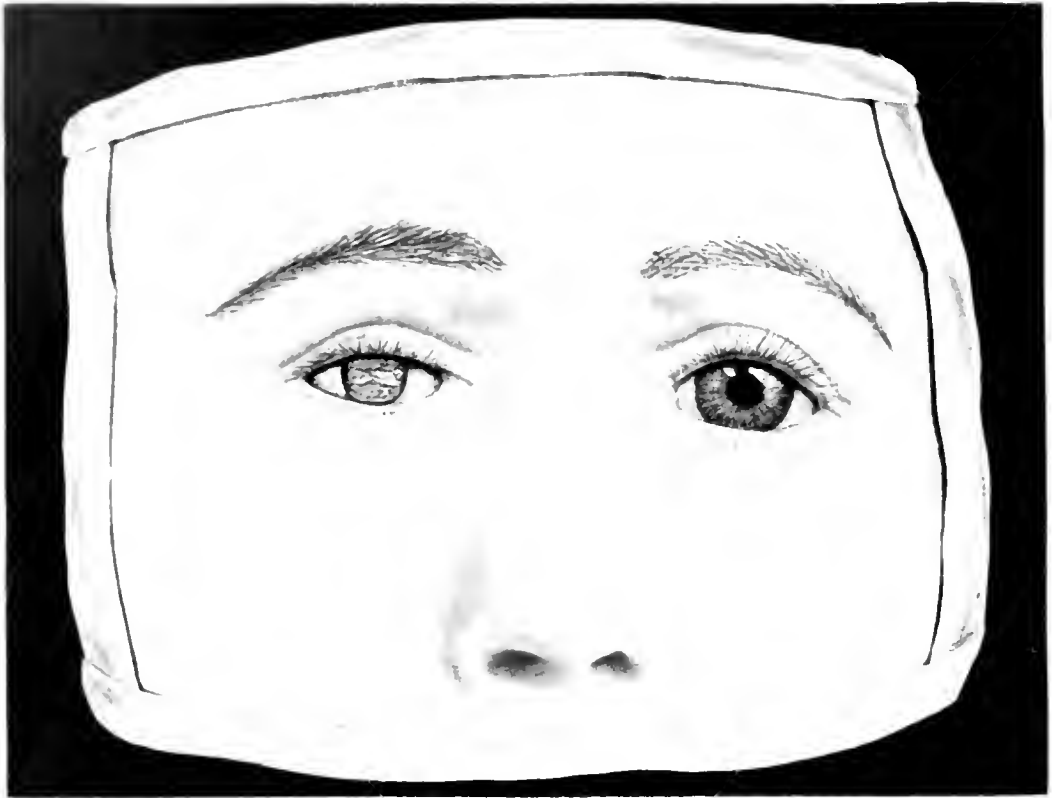


Fig. 36. Phthisis bulbi incipiens.
Adhesion form of Keratitis.

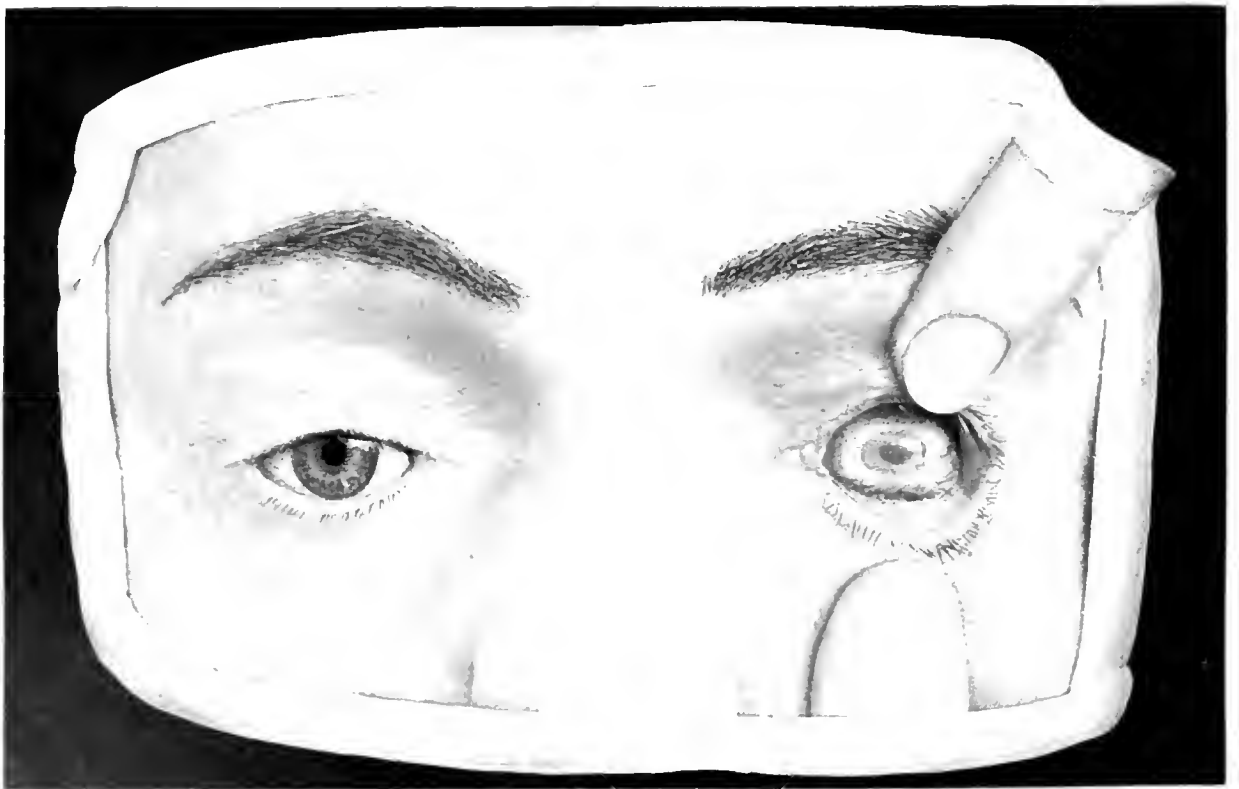


Fig. 37.
Phthisis bulbi quadrata.

Gonorrhoeal Conjunctivitis. *R. H.*

Plate XXIV, FIGS. 34, 35; Plate XXV, FIGS. 36, 37; Plate XXVI, FIG. 38

Gonorrhoeal diseases of the eye are usually due to contact with an external source of infection, but they may also be produced endogenously, so that they may be divided into **contact** and **metastatic** gonorrhoea of the eye. Metastases may likewise take place into other parts of the body from an infection of the eye, particularly into the joints.

Contact gonorrhoea of the eye is subdivided into the gonorrhoeal conjunctivitis of infants, or ophthalmia neonatorum, and of adults, but the only differences between them are the way in which they originate and the prognosis.

OPHTHALMIA NEONATORUM

Gonorrhoeal disease of the eye soon after birth is always due to a contact infection. *Neisser's* gonococci are enabled to enter the conjunctival sac during labor, or, in very many cases, after birth through a fault in cleansing. The disease appears after a short period of incubation, which varies from a few hours to several days, in proportion to the severity of the infection, but is usually 2 or 3 days, less often 4 or 5. The lids become very red and swollen, chemosis is rarely present, while a secretion, that is at first turbid and later pure pus, escapes from the palpebral fissure. Gonococci, which do not take *Gram's stain*, are usually to be found in great numbers, lying in pairs within the leucocytes or the desquamated epithelium in the pus. After some weeks the thick, creamy pus becomes thinner and finally ceases, never leaving a chronic conjunctivitis.

When the pus stagnates it is very apt to corrode the epithelium of the cornea and to cause the tissue to break down more or less. The defect thus produced is covered by an opaque, whitish cicatrix, called a leucoma, after the inflammation has run its course. In Fig. 35, Plate XXIV, a little black mark can be seen in the upper part of the leucoma, indicating the presence of an anterior synechia.

A small perforation took place in the cornea, through which a bit of the iris prolapsed, where it was caught and firmly held during the process of healing. (See also Fig. 55, Plate XXXVIII, which shows a leucoma of the entire cornea. In this case the cornea was destroyed by small pox, but the appearance is just the same when the leucoma is due to gonorrhoeal conjunctivitis.) Sometimes the entire cornea breaks down into pus, so that the iris is laid bare (see Plate XXVI, Fig. 38). The margins of the pupil become drawn together, the surface cicatrized over, and the wall thus created is usually driven forward by the intraocular pressure so as to form a total staphyloma of the cornea (see Plate XLI, Fig. 59).

When the pus finds its way into the interior of the eye it excites a long, severe inflammation which is followed by gradual atrophy of the eyeball, phthisis bulbi (see Plate XXV, Figs. 36 and 37).

During the attack the conjunctiva is very red and swollen, and the papillae spring up in ridges like cockcombs, but (no such follicles as are seen in follicular conjunctivitis and trachoma are ever produced.) No scars are left in the conjunctiva after the disease has run its course.

Fig. 55. 148.

GONORRHOEAL CONJUNCTIVITIS OF ADULTS

Statistics might seem at first to support the view that the adult eye is but slightly sensitive to the gonococcal virus, for gonorrhoeal urethritis is very common in large cities, while the percentage of these patients in whom the eye becomes infected is very small, perhaps not one in a thousand, but the evidence is very convincing that this assumption is not true. The eye escapes because it is too well protected to be infected easily, and gonococci are delicate organisms that are easily killed. The fresh pus must be brought directly into the eye, and even rubbing with an infected hand does not always suffice, because we are not apt to rub the conjunctiva, and the gonococci are not able to attack the skin of the lid. We have quite enough examples to prove that the eye is very sensitive when the virus is introduced.

Gonorrhoeal conjunctivitis starts in much more violently in the eye of an adult than in that of an infant, a fact that it is important to remember. Many patients who have gonorrhoea, and know that the disease may be transferred to the eye, are constantly worried. If a few red blood vessels appear in the conjunctiva their anxiety naturally increases and they are apt to come to the office. When the conjunctiva is very red we can usually tell from its duration whether

the inflammation is gonorrhoeal or not. The symptoms of gonorrhoeal conjunctivitis begin very violently with lachrymation and increase from hour to hour, so that although the tears may be clear in the morning, they are turbid by noon, and by evening there is a stream of pus from the eye. Hence, when we learn that the symptoms were present yesterday, or the day before, and when the characteristic picture is absent, we may reassure the patient, for the infection is not gonococcal. The characteristic picture of gonorrhoeal conjunctivitis presents a great oedematous swelling of the lids, chemosis of the bulbar conjunctiva, and, above all else, a constant stream of pus from the eye.

The cornea is so much more apt to be involved in adults than in infants that we are not always able to save it, even with the most careful treatment.

METASTATIC GONORRHOEAL CONJUNCTIVITIS

Metastases into the conjunctiva, just as into the joints, are not uncommon in gonorrhoea, and they have been observed rather often in modern times. The eye, indeed, seems to be predisposed to them.

The clinical picture is quite different from that produced by contact infection. There is no great suppuration, and we see simply a conjunctivitis with some oedema in its vicinity, often associated with an iritis that presents no distinguishing characteristics. No gonococci can be found in the secretion in the great majority of cases. Probably we are dealing here with a mixed infection, for we know that gonococci do not excite such a plastic inflammation as is often present in this form of iritis, but many of the symptoms shown in gonorrhoeal arthritis are exhibited in the eye.

Prognosis of Gonorrhoeal conjunctivitis

The prognosis of ophthalmia neonatorum is not bad under good treatment. It is almost always possible to save the eyes undamaged when proper treatment is begun in time and maintained rigorously, but in the absence of such treatment the pus is apt to erode and destroy the cornea to a greater or less degree.

The prognosis is not as good in adults, for it is often impossible to save the cornea, even by our best efforts.

Prophylaxis is most important. Infants should be bathed as soon as born, but the water of the bath should never be allowed to come in contact with the eyes. These should be washed with clean

water in a separate basin. Then, following *Crede's* method, a drop of a 2% solution of silver nitrate should be instilled in each eye, or it may be better to use a weaker solution, one of about $\frac{1}{4}\%$.

Adults who have gonorrhoea should be warned not to touch their eyes, [or those of other people,] and should be instructed to wash their hands with soap and water whenever they have come in contact with the pus. When only one eye is affected the other should be covered by a shield made of a watch glass and adhesive plaster.

Treatment

[The main reliance in the treatment of gonorrhoeal conjunctivitis due to external infection is cleanliness. In both infants and adults the conjunctival sac should be irrigated as often as is necessary to keep the eye fairly free from pus, the intervals ranging from every 15 minutes to every hour. Saline is as good a solution as any for the purpose, for antiseptics are too apt to do harm.] During the irrigation the eye must not be wiped or touched, as the cornea may readily be injured in this way, especially by unskillful hands, and such an accident is very dangerous. The fluid should simply be allowed to flow over the eye in the palpebral fissure from some wet cotton, or from a bottle with an aperture in its stopper, while the patient opens and closes his eyes. All of the pus may be removed in this way.

The best drug to use is **silver nitrate**, for which some substitute protargol or argentamin. The classical method of *v. Graefe* is to evert the lids and paint them with a 3% solution every day, but I prefer to wash the pus frequently from the eye with a much weaker solution, $\frac{1}{3}$ to $\frac{1}{10}\%$.

[During the intervals between the irrigations it is well to apply ice cloths continuously, changing them before they become warm, for, although the coolness produced does not extend to the conjunctiva, they reduce the swelling of the lids and alleviate the pain. It is better not to use them on weak and anaemic patients, and ice must never be applied, as it may cause necrosis of the skin.]

Many authors recommend a daily application of lenicet or blennolenicet ointment, or of euvaseline, as a means to guard against the maceration of the cornea by the pus, [but nothing is yet known that will do this with certainty.

Serum treatment has not yet proved very successful, but its advocates dissolve gonococcus serum in the water used for irrigation and dust powdered serum into the eye immediately afterward.]

We are impotent as regards the formation of a leucoma when the cornea becomes involved. [We may substitute hot for cold applications, in order to promote the nutrition of the cornea, we may apply leeches to the temple when the inflammation and oedema is very great, we may scarify the conjunctiva in adults to disburden it, we may perform canthotomy to relieve the pressure of the lid upon the cornea, and we may instill atropine to control the iritis, but the final result remains uncertain. Some apparently bad cases recover, but usually a leucoma is formed.] When this is partial and lies in the center of the cornea, an artificial pupil may be made by excising a piece of iris at its side, performing an optical iridectomy (see Plate XXIV, Fig. 35). The white spot may be tattooed with india ink after all signs of irritation have disappeared, in order to improve the cosmetic appearance.

[The only treatment needed for metastatic gonorrhoeal conjunctivitis is rest in bed, bathing the eye with some bland lotion, like a boric acid solution, and atropine to control the iritis if present.]

Pinguecula. Pterygium.

Plate XXVI, FIG. 39

A **pinguecula** is a more or less triangular elevation of the bulbar conjunctiva on either side of the cornea, in the region of the palpebral fissure, which gradually assumes a straw yellow color. It is a senile change, and its development is favored by exposure to inclement weather, smoke, dust, or a hot, sunny climate. It was formerly thought to be composed of fat, as its name implies, but it really consists of an increase of the elastic tissue with a granular pigment.

A pinguecula is of no importance, except for the disfigurement it may occasion, and the fact that it may form the starting point of a pterygium, but its appearance sometimes frightens the patient, or his relatives, so its nature should be understood.

No treatment is needed. [If the patient desires to have it removed to improve the cosmetic appearance, it may be excised, or destroyed with the actual cautery.]

PTERYGIUM

A **pterygium** is a triangular fold of conjunctiva which grows horizontally over the cornea from its inner or outer side. Its rounded apex, lying on and firmly attached to the transparent cornea, toward the center of which it is directed, is called the head; its neck is the thinner portion extending from the head to the body and attached along its middle to the cornea; its broad body lies on the sclera.

Origin and course

A pterygium originates in a pinguecula, which suddenly starts to grow over the cornea, dragging after it a fold of conjunctiva. It advances very slowly, producing no inflammation, and in the course of years may reach or pass beyond the center of the cornea. It is very vascular, [the lines of the bright red blood vessels give it a certain resemblance to the wing of a fly.] occurs chiefly in elderly people, and is more common in hot than in temperate climates.

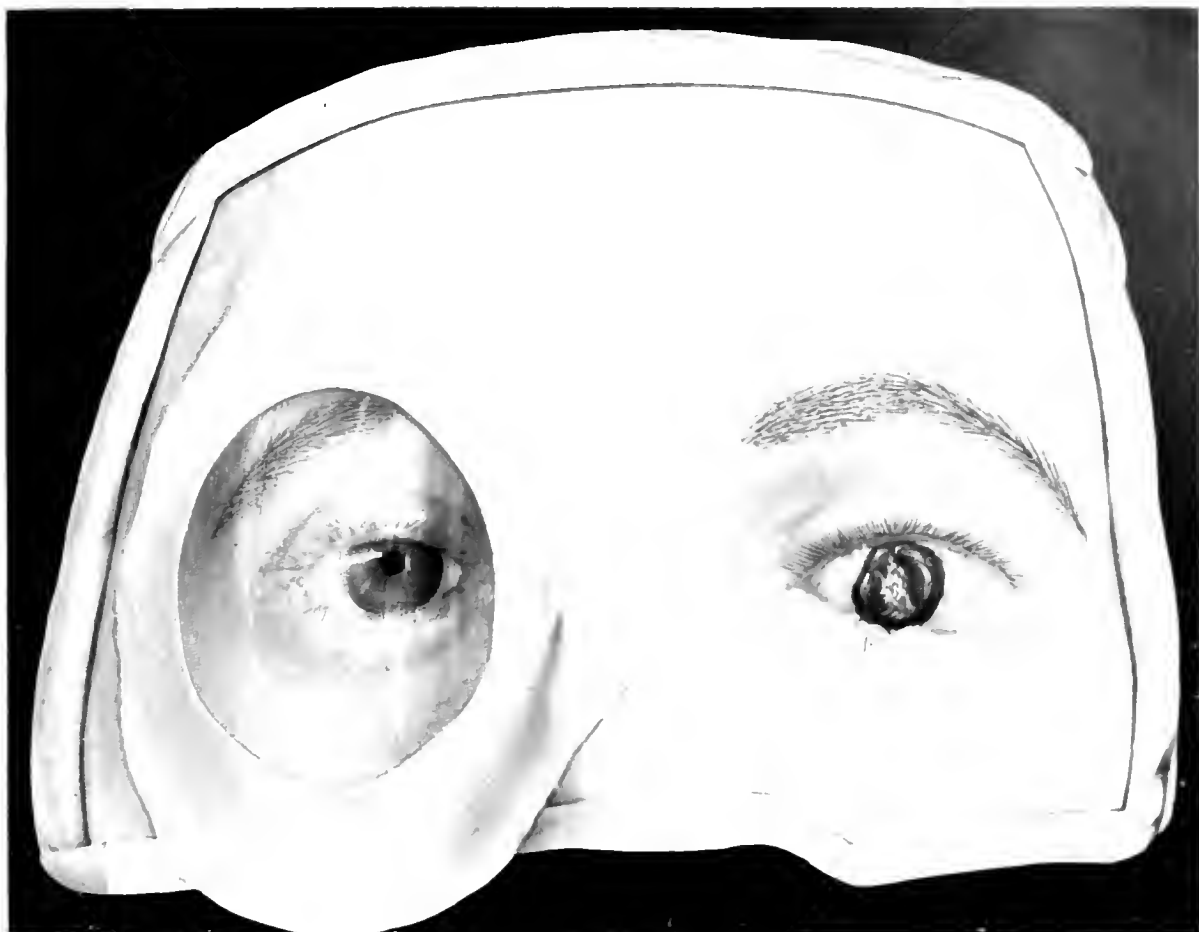


Fig. 38.
Conjunctivitis gonorrhoeica adultorum.
Prolapsus iridis totalis.

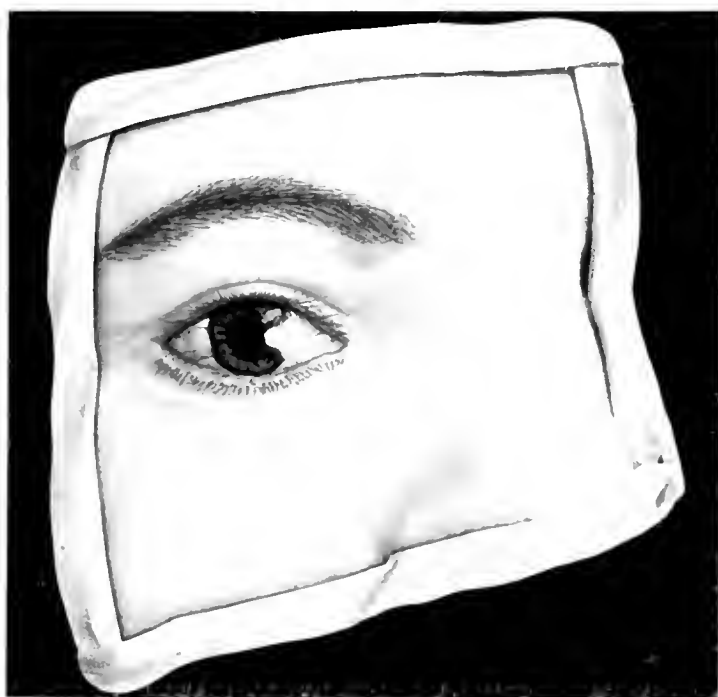


Fig. 39.
Pterygium und Pinguecula.

Prognosis

The prognosis is not good, unless the growth is removed by an operation. The vision begins to suffer as soon as the apex encroaches on the area of the pupil, and becomes reduced to perception of light when this area has been completely covered.

Treatment

Treatment consists of ablation of the growth and closure of the defect left in the conjunctiva with sutures. This should be done as early as possible, for the surface of the portion of cornea affected never again becomes smooth, so that some impairment of vision will always remain after the removal of a far advanced pterygium.

[The simplest operation is to cocaine the eye, seize the neck near the limbus with forceps, pass a Graefe knife or a keratome beneath it, gradually work the knife toward the center of the cornea so as to strip off the pterygium until its head is completely freed, and then to make two convergent incisions through the body, so as to remove a rhomboidal piece of the growth. The upper and lower edges of the defect thus formed in the conjunctiva are brought together with sutures. Relapses are rather common after this operation and the following one is to be preferred.

In *McReynolds'* operation the pterygium is separated from the cornea in the manner above described, and undermined to its base. An incision is then made through the conjunctiva along the lower margin of the body from its neck to its base, and the conjunctiva below the wound is undermined so as to form a pouch. A mattress suture of catgut is passed through the head of the pterygium, leaving a loop on its inner surface, the two needles of this suture are then passed into the pouch and out through the conjunctiva well down toward the transition fold. The pterygium is then drawn down into the pouch and the suture tied. The eye should then be bandaged for 4 or 5 days.

When the head of the pterygium is very broad *Knapp's* operation may be preferred. After the head and neck have been detached from the cornea the pterygium is split and each portion is secured in an incision in the conjunctiva made upward and downward from the body about 4 mm. from and parallel to the margin of the cornea. This leaves a defect that needs to be covered by sliding together two conjunctival flaps, one from above, the other from below, and suturing them.]

Diphtheritic Conjunctivitis.

Plate XXVII, FIG. 40

Diphtheritic conjunctivitis is the name applied to the inflammation of the conjunctiva produced by the Klebs-Loeffler bacillus. The malignant, deep, necrotic form, in which the cornea is apt to be involved, was the only one with which *v. Graefe* was acquainted, but soon after his time a mild, superficial form was described and carefully differentiated from diphtheria as croup. This distinction prevailed until *Babes* in 1886 demonstrated the diphtheria bacillus to be the active agent in diphtheritic conjunctivitis, and the later discovery that this bacillus is present in both severe and mild cases, which changed our views. Both forms are included to-day under the one name.

Whenever we have to deal with a conjunctivitis that presents a superficial pseudomembrane, we suspect it at once to be a case of diphtheria, but we can only suspect, because the clinical diagnosis cannot be positive in the absence of a bacteriological examination. Gonococci, staphylococci, and more commonly streptococci likewise give rise to the formation of pseudomembranes, while on the other hand, virulent diphtheria bacilli may excite a simple catarrhal conjunctivitis with no pseudomembrane. Why the same bacilli should excite a superficial, benign disease in one case, and a deep, necrotic one in another, we do not know.

Ordinarily the diphtheria bacilli are not present in pure cultures, but are intermixed with staphylococci, streptococci, and sometimes with avirulent xerosis bacilli, the so-called pseudodiphtheria bacilli, but these admixtures seem to exert no influence on the course of the disease. [Some writers believe the corneal complications to be mainly due to the pus germs rather than to the diphtheria bacilli, and it is certain that serum treatment often fails to influence the corneal complications even when it is of marked benefit to the conjunctival trouble.]

Diagnosis

The diagnosis in mild cases cannot be made except by the demonstration of Loeffler's bacilli.

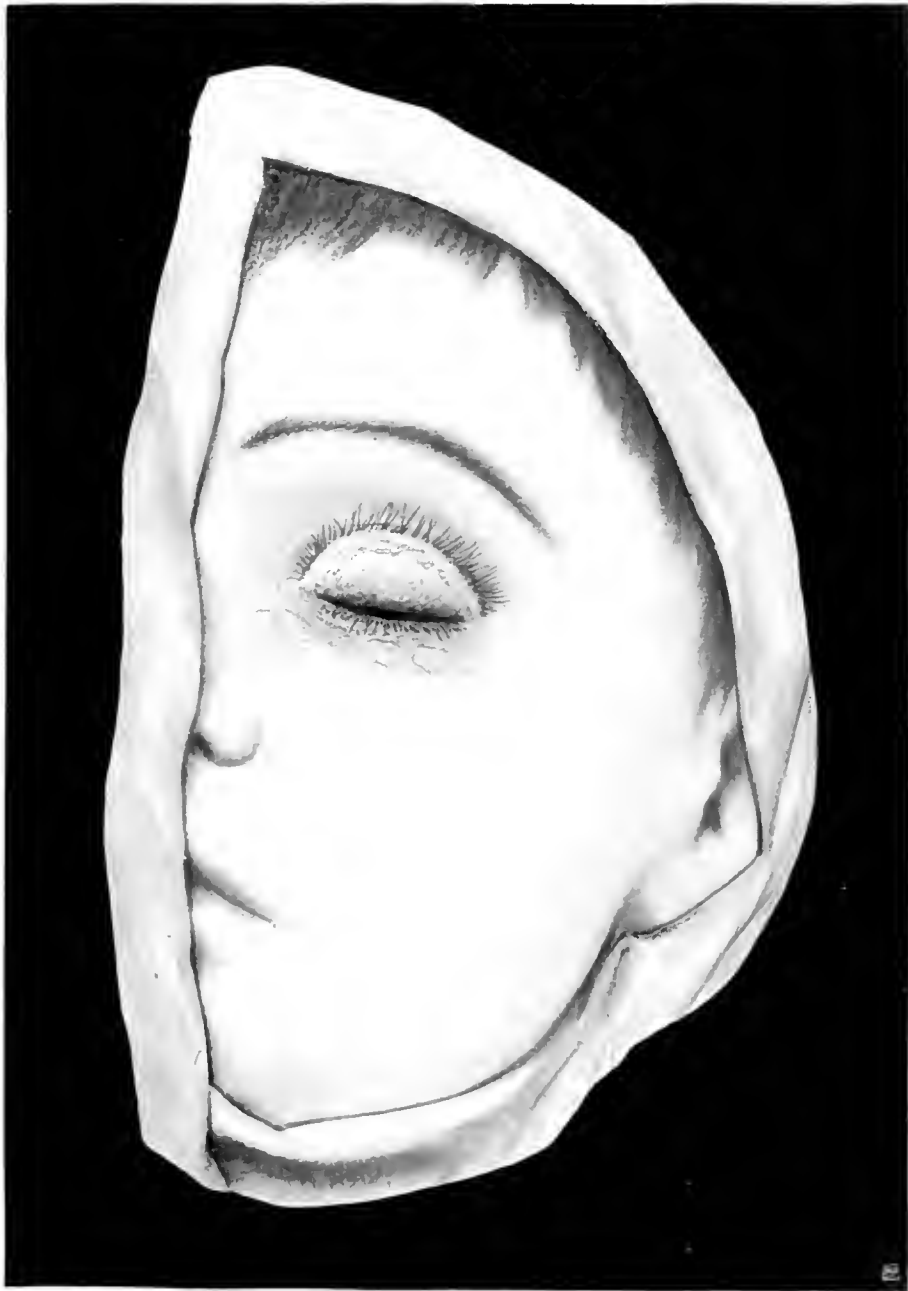


Fig. 40.
Conjunctivitis diphtherica.

Treatment
Prognosis

The prognosis is always doubtful. Many patients recover, but some cases that seem to be running a mild course, suddenly become malignant, destroy the cornea, induce gangrene of the lid, and threaten life through a general infection.

Treatment

Antitoxin should be injected subcutaneously in large doses. [This should be done as soon as the disease is suspected, without waiting for proof, unless it can be obtained immediately, for the earlier in the disease antitoxin is administered the more certain will be a good result, and delay is dangerous.] In addition the conjunctiva should be cleansed frequently with a 1 to 5000 solution of either sublimate, or potassium permanganate.

The false membranes should be removed only when they can be detached by a very gentle stroking; all violence must be carefully avoided.

[The same precautions to prevent the spread of the disease must be observed that are practiced in other forms of diphtheria. The patient must be isolated, and it is well to immunize the other members of the family.]

Phlyctenular Conjunctivitis and Keratitis.

Plate XXVIII, FIG. 41; Plate XXIX, FIG. 42; Plate XXXII, FIG. 47

Phlyctenular disease of the eye, also known as eczematous, scrofulous, and lymphatic conjunctivitis and keratitis, sometimes as superficial keratitis, is characterized by the formation of globular projections, of about the size of millet seeds, on the surface of the eyeball. These are called *phlyctenulae*, or eczematous efflorescences, and occur most often in rows along the corneoscleral margin, or limbus (see Plate XXIX, Fig. 42). A triangle of bloodvessels soon appears in the conjunctiva with the phlyctenule at its apex. Sometimes the ulcer formed by the phlyctenule at the limbus progresses into the cornea, drawing after it a leash of blood vessels (see Plate XXXII, Fig. 47). This is called *fascicular keratitis*.

After a short time the apex of the phlyctenule is cast off, leaving a small, gray ulcer, which may soon become clean, covered with epithelium and heal, the entire course occupying only a few days.

Eczema is to be found elsewhere on the head or face, in the majority of cases, and usually the fundamental cause is scrofulosis.

Prognosis

The prognosis is good. The individual phlyctenules run their courses rapidly and surely, but recurrences are very apt to take place so long as the fundamental disease is not treated. When they persist for some time in the cornea they cause infiltrations and ulcers, which leave scars of more or less density when they heal, maculae corneae.

Treatment

Treatment is to be directed first of all to the general condition. All of the hygienic measures described under the constitutional treatment of eczema of the margin of the lid, diet, iron, cold baths, perhaps



Fig. 41.
Blepharitis and Conjunctivitis eczematosa.

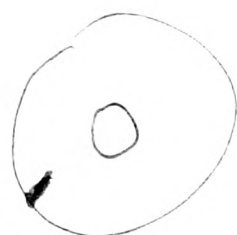




Fig. 42.
Conjunctivitis and Keratitis phlyctaeulosa
Eczema faciei.

sun baths, and fresh air, are indicated. As constipation is frequently present it is well to begin with a dose of calomel. The eczema on the scalp, face, nose, or ears, must be cured; otherwise there is danger of relapse. Yellow oxide ointment, 1% of yellow oxide of mercury in vaseline, should be applied to the eye daily and rubbed in with the lid. In the later stages very fine calomel may be dusted into the eye for the purpose of clearing up the corneal opacities.

[Children with phlyctenular keratitis are very apt to suffer from photophobia and blepharospasm, the degrees of which are not proportioned to the severity of the disease. These can often be relieved by douching the eyes with cold water, sometimes permanently, more often not. A drop of a solution of holocain will give temporary relief from the photophobia, but the use of cocaine for this purpose is inadvisable. Above all, the eye must never be tied up; air and light should be allowed to enter, as they are beneficial. The little patients must not be permitted to bury their faces in pillows, or to cower in dark corners. Colored glasses give considerable relief when worn in the sunlight, out of doors.

The abundant lachrymation is apt to cause a dermatitis, particularly at the outer canthus, with little fissures or excoriations that are very painful and excite an intense blepharospasm. Such excoriations must be dealt with before efficient treatment can be given to the eye. The skin should be dried with cotton, silver nitrate or bluestone applied to the sores, and then the skin should be protected from the constant wetting by the application of a suitable ointment. In very bad and obstinate cases of blepharospasm a cantholysis is advisable.

When the cornea is involved atropine should be instilled and hot compresses applied at regular intervals. One drop of a $\frac{1}{2}\%$ solution of atropine three times a day, and hot compresses changed very frequently for ten minutes every two hours, is about right for the average case. When the ulcer begins to improve its progress may be hastened by the use of yellow oxide ointment.

The progress of a fascicular keratitis may often be cut short by touching the advancing edge with a fine hot cautery point, with tincture of iodine, or with pure carbolic acid. Yellow oxide ointment should then be applied.]

Vernal Conjunctivitis.

Plate XXX, Figs. 43, 44

Vernal conjunctivitis, or vernal catarrh, is a rather rare disease, met with principally in boys, the chief subjective symptom of which is a very troublesome itching and burning of the eyes. It begins in the spring, affects both eyes as a rule, disappears, or becomes milder, after a few months, and recurs regularly for quite a number of years.

The disease presents two very characteristic **pathological changes**, one on the conjunctival surface of the tarsus of the upper lid, the other at the limbus of the cornea. It is not necessary that both should be found in any given case, and many authors make a distinction between a palpebral and a bulbar form, yet the two occur together in typical cases. It may also happen that both are absent, and that the conjunctiva may exhibit simply an injection, and a slight, uniform thickening; in such a case the diagnosis can be made only from the history.

The **palpebral** change consists of broad, flat excrescences scattered over the tarsal conjunctiva, which, when placed close together, cause it to look as though it were covered with paving stones. These excrescences often are several millimeters broad, not as thick, round at the periphery or rendered angular by the pressure of adjoining growths, and have a flat or somewhat concave surface. *Horner* has aptly compared them to mushrooms. They are hard, sometimes of a cartilaginous consistence. The entire conjunctiva is pale rather than inflamed, while over and about the excrescences it has a peculiar, bluish white, milky appearance. Sometimes the "paving stones" are present in large numbers and lie close together, sometimes they are quite isolated; they may look as though they were worn away and their original limits may be indicated only by delicate furrows.

The conjunctiva of the lower lid usually is only thickened, without folds, but it presents the same pale, milky appearance. There are no changes in the transition folds, no follicles, and no severe inflammation.



Fig. 43. Conjunctivitis vernalis.
Changes in the Conjunctiva.

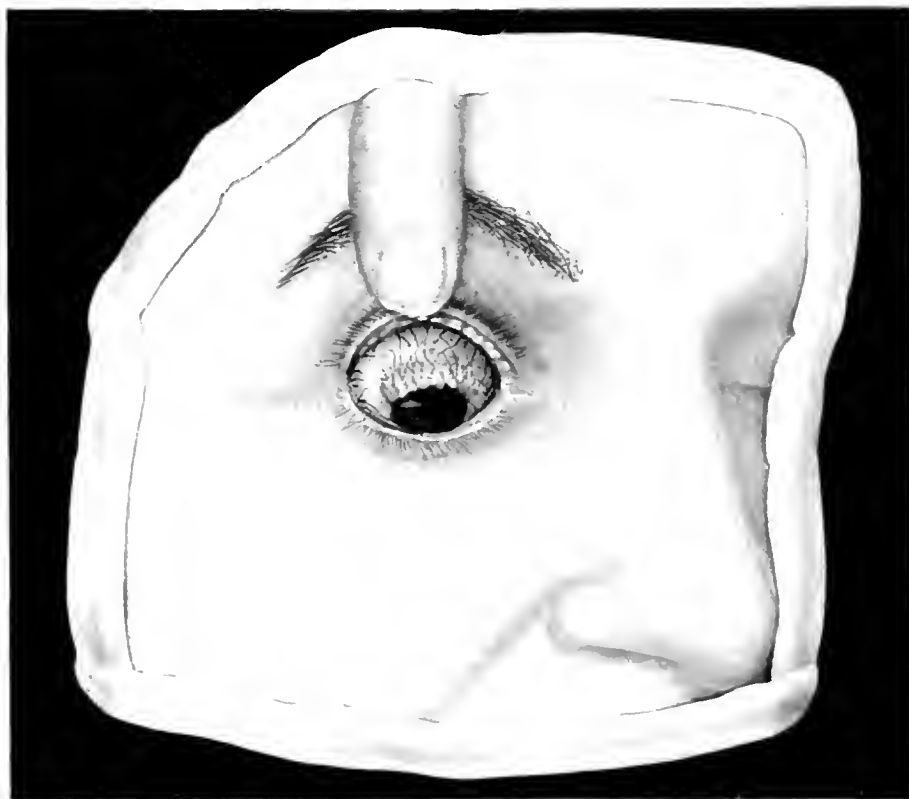


Fig. 44. Conjunctivitis vernalis. Changes in the Limbus.

The change in the **bulbar conjunctiva** is still more marked and is apt to attract attention sooner. Some vessels may be seen extending from the periphery to the limbus at the site of the pinguecula. At the limbus are uneven, hard prominences of a bluish white color, which may be confined to the inner or outer margin of the cornea, or may form a narrow, gelatinous looking band about its entire circumference. Sometimes these elevations are flat and remain so for years, in other cases they develop into uneven nodules.

Pathology

Pathologically the condition is one not of conjunctivitis, but of a purely hypertrophic process at the affected places. The bluish, milky lustre of the conjunctiva is due to the great thickening of its epithelium, which is three times as thick as normal over the excrescences, so that 30 layers of cells often are superimposed one on another. The epithelium also sends down deep shoots that form nests and other figures, so as to produce a picture which resembles that of carcinoma, but can be distinguished from the latter by the fact that these shoots do not penetrate the deeper layers. [Proliferation also takes place throughout the stroma of the conjunctiva and in the subepithelial connective tissue. The vessels likewise are affected. A peculiar, stringy secretion may often be found in the transition folds. It is not usually free, and sometimes can be found only after considerable irrigation.]

Diagnosis

This disease is sometimes mistaken for trachoma, but its true character should be recognized from the density of the palpebral excrescences, their resemblance to paving stones, and the milky appearance of the conjunctiva. No such mistake should be made when the characteristic excrescences at the limbus are also present. [The bulbar excrescences need at times to be differentiated from phlyctennules. The latter develop rapidly, soon break down and are absorbed, while in vernal catarrh the prominences are hard, firm, and never break down.]

Prognosis

The prognosis is good, in so far that recovery eventually takes place without damage to the cornea, but not good as to its duration, for we have no means at our disposal with which to shorten its course.

Treatment

For treatment only such slightly astringent collyria as are used in catarrhal conjunctivitis are to be recommended. Cauterization of the affected parts makes matters worse. Xeroform or anaesthesin may be dusted into the eye to relieve the itching, or a 1 to 1000 solution of dilute acetic acid may be instilled several times a day. Massage with a 2 to 5% ointment of yellow oxide of mercury may be employed for the proliferations at the limbus.

When the papillary proliferations become very large they may be cut off with scissors, or removed with the wire loop of an electric cautery, [but this does not prevent recurrence. *Fox* strongly recommends grattage, from which he claims to have obtained surprisingly good results.

Quite a feeling of relief can be obtained frequently by irrigation of the fornices with a solution of boric acid to remove the long threads of secretion from the transition folds.

It has been demonstrated that the symptoms ameliorate when light is excluded from the eyes, and also when an appliance is worn that excludes air while vision is permitted through a plain glass, but they return at once when the patient goes out of doors with bare eyes. Some authors think it due to the ultraviolet rays, so euphos, or Hallauer's, glasses are sometimes prescribed. Others think it related to the sunlight dermatoses, but these seldom appear in the spring.]

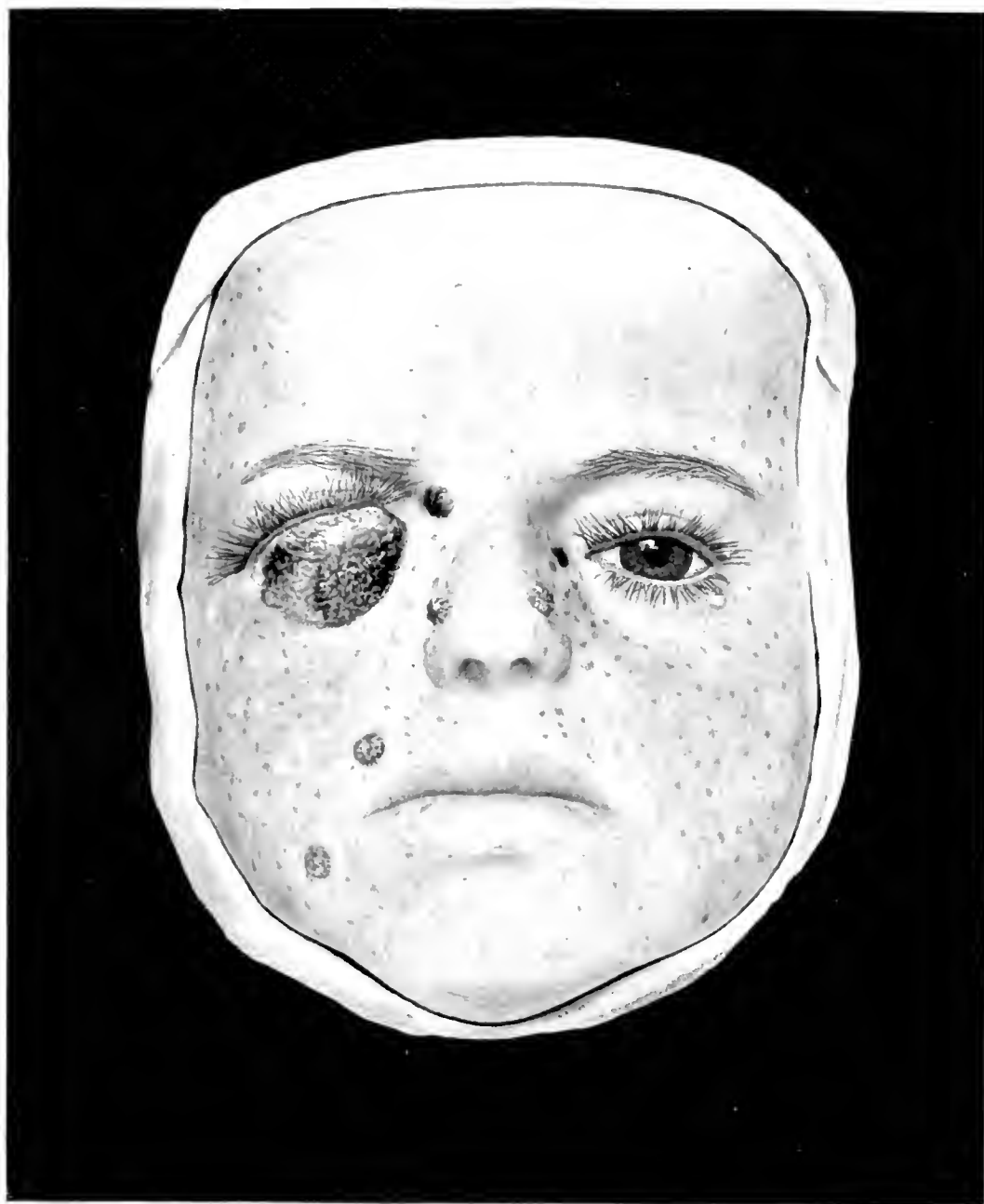


Fig. 15.
Xeroderma pigmentosum. Tumor epibulbaris.

Epibulbar Tumors. Xeroderma Pigmentosum.

Plate XXXI, FIG. 45

Epibulbar tumors occur chiefly in middle aged and elderly persons, and start almost invariably from the corneoscleral margin, very rarely from the bulbar conjunctiva. According to *Virchow* strongly pigmented connective tissue cells occasionally appear at the margin of the cornea, and these may coalesce to form little, black spots, known as melanomata, which are frequently the starting points of tumors. In some cases these tumors seem to result from an insignificant traumatism, in others they appear on a scar, while a constant irritation, such as is produced by working in dust, seems to favor their development, but in very many no cause for their growth can be assigned. The first thing perceived, as a rule, is a spot of pigment at the limbus which grows slowly into a tumor, either a sarcoma or a carcinoma.

A **sarcoma** is apt to have a more slender trunk, to spread more superficially, to be soft and very vascular, and to look like a more or less pigmented, reddish knob.

A **carcinoma** is broader, [its surface is less smooth, its color more inclined to white, it is more likely to lack pigment, and less apt to bleed, even when ulcerated.] Often a careful examination with a probe will show it to be simply lying on the surface of the cornea, even when at first glance it appears to be adherent. It grows fast to the conjunctiva, and when it can be moved with the latter over the surface of the sclera we know that it has not penetrated deeply.

If a neoplasm that starts in this manner is not removed it grows slowly, invades the conjunctiva and cornea, and may finally develop into a colossal tumor.

The patients come under observation as a rule when these more or less pigmented, fungus-like tumors are circumscribed and about as large as peas or beans. At this time a thorough removal may prove sufficient, especially when the growth is confined to the most

superficial layers. The tumor should be cut away with a knife, or with scissors, and then its base must first be curetted thoroughly with a sharp spoon and then cauterized with a *Paquelin* or electric cauter. [When the removal of the tumor leaves a large gap in the conjunctiva the defect must be repaired by bringing the edges of the wound together, by undermining and sliding flaps of conjunctiva, or by the transplantation of a graft of mucous membrane from the lip or elsewhere. The x-rays may subsequently be used.] In some cases this operation suffices to effect a permanent cure, but more often a recurrence takes place that grows very slowly. The surgeon should not fail to call the patient's attention to the malignancy of these tumors and to urge upon him the need that he should return from time to time for examination.

When the tumor is large and has invaded deeper tissues the eye must be sacrificed, and the entire orbit should be exenterated under certain circumstances, but in many of these cases metastases have already taken place and the patients die of exhaustion, or from tumors in other organs.

XERODERMA PIGMENTOSUM

Xeroderma pigmentosum is a rare and peculiar disease that was described first by *Kaposi* in 1870. The total number of cases on record does not much exceed 100, which is a small number, especially as the cases are generally multiple when found, several members of a family being affected, and as the disease excites so much interest that a case can scarcely escape publication. Some cases have been reported several times by different authors.

There seems to be a congenital predisposition to this disease, for it has been observed in several children of the same family almost every time, once in 7 brothers. Sometimes only the children of the same sex were affected, in other families the children of both sexes, just as happens in other hereditary diseases. I have seen the disease in two brothers, in the elder of whom it was far advanced. But in none of the cases reported did the parents have any disease that could be determined with certainty to have a causal connection with that of the children; there was no consanguinity, no general or cutaneous disease, or debility. The children were born with normal skin, but during the first, or, at the latest, the second year of life circumscribed red spots appeared on the face, neck, hands, or wherever the skin was exposed to the influence of the rays of the sun. These spots

disappeared with a little desquamation after a short time, but reappeared after each exposure to the sun.

Lukasiewicz noted a diffuse redness of the skin as the earliest sign of the disease. As often as his little patient was taken out of doors on a sunny day, a diffuse redness and swelling would appear in the exposed parts of the skin a few hours later. The redness paled under the pressure of the finger, and the pressure was painful. The patient was distinctly depressed, but there was no rise of temperature. These symptoms would increase for a few days, and then pass away with a slight degeneration. This was repeated frequently during the spring and summer, but no erythema was seen during the winter while the child was kept in the house.

Permanent changes appear very soon in typical cases. Pigmentation takes place in what may be called the second stage. Numerous spots of pigment, resembling freckles, are found on the parts of the body exposed to the light, while the pigment of the skin between them diminishes to such an extent as to create in many cases large, white patches that are totally destitute of pigment. Generally the pigmentation soon predominates so that the affected portions of the skin appear to be spotted with brown or black.

The next step in the course of the disease is the development of numerous vascular dilatations, usually in the form of small, superficial teleangiectases, more rarely of angiomatous tumors.

The skin becomes atrophic, smooth, and loses its normal furrows and folds. When a piece of it is examined microscopically an atrophy, a thinning and flattening of the papillae and epidermis is found, resembling that produced by senile degeneration. We find an atypical outgrowth of the cones of the rete mucosum in the chronically changed cutis, hyperplasia of the sebaceous glands, and ectasias of single vessels (*Lukasiewicz*).

Thus the conditions are provided for the final stage of the disease, the formation of multiple carcinomata. Wart-like growths develop here and there, usually at places which correspond to folds in the skin, so that the brown-black parts of the body are quite covered with them in many cases. True epitheliomata develop from a greater or less number of these nodules and follow the usual course of these cancers. They progress and break down the tissues into large ulcers, or cause death through a gradually increasing cachexia, apparently without metastases into the internal organs.

Subconjunctival Lipoma.

Plate XXXII, FIG. 46

Subconjunctival lipoma is a congenital tumor of a yellow color, as seen through the conjunctiva, not so very rarely to be found at the outer canthus, between the insertions of the external and superior recti. It is freely movable over the sclera with the conjunctiva, which is somewhat thickened over its surface. It has an abrupt or undermined margin toward the cornea, and slopes away from this gradually toward the temple into the orbital fat. Ordinarily it is about as large as a pea or a bean, and then it cannot be seen except when the eye is turned toward the nose, but when large it can be seen while the gaze is directed straight forward.

Under the microscope the tumor is seen to be composed essentially of hyperplastic fat tissue.

Diagnosis

It is difficult to make an error in diagnosis, as other forms of tumors do not occur in this region.

Prognosis

The prognosis is perfectly good. The tumor may begin to grow at the time of puberty, yet it always remains benign. The only harm it can do is to prevent an exact closure of the lids by protruding between them.

Treatment

The tumor may be left alone when it is small, though it is better to remove it because of the disfigurement it may occasion. The removal of the mass of fat after an incision through the conjunctiva is simple and does not need to be radical. [Sometimes the conjunctiva is found to be so firmly adherent as to render necessary the removal of a portion of it together with the tumor. Too thorough an extirpation may give rise to the danger that the cicatrix will limit the inward movement of the eye.]

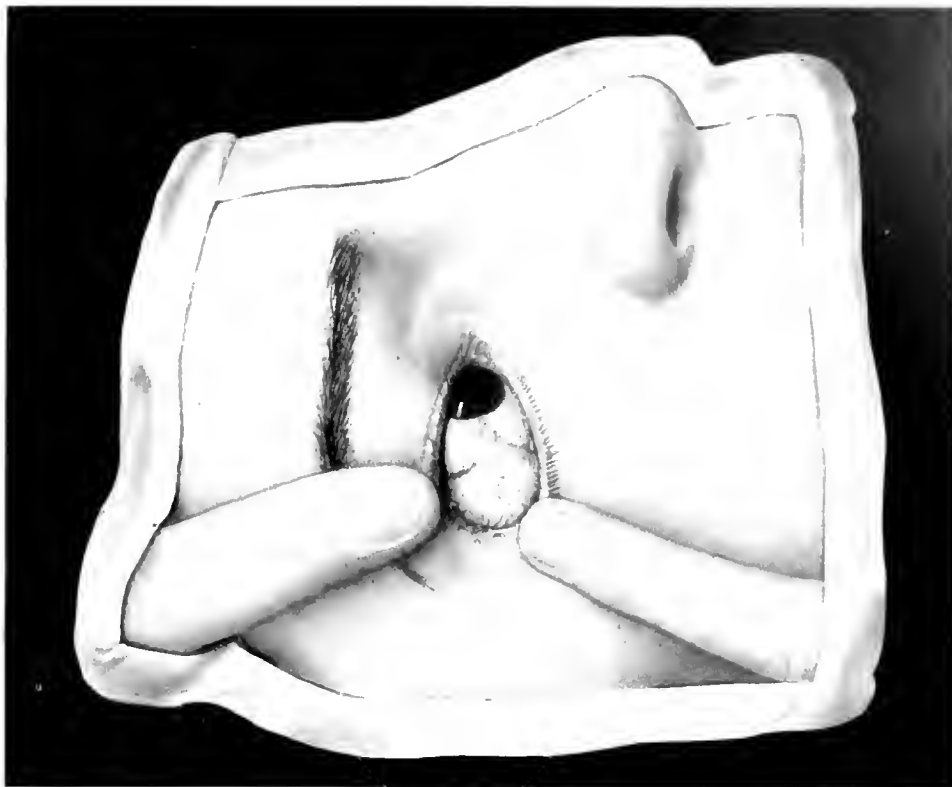


Fig. 46.
Lipoma subconjunctivale congenitum



Fig. 47.
Keratitis fascicularis.

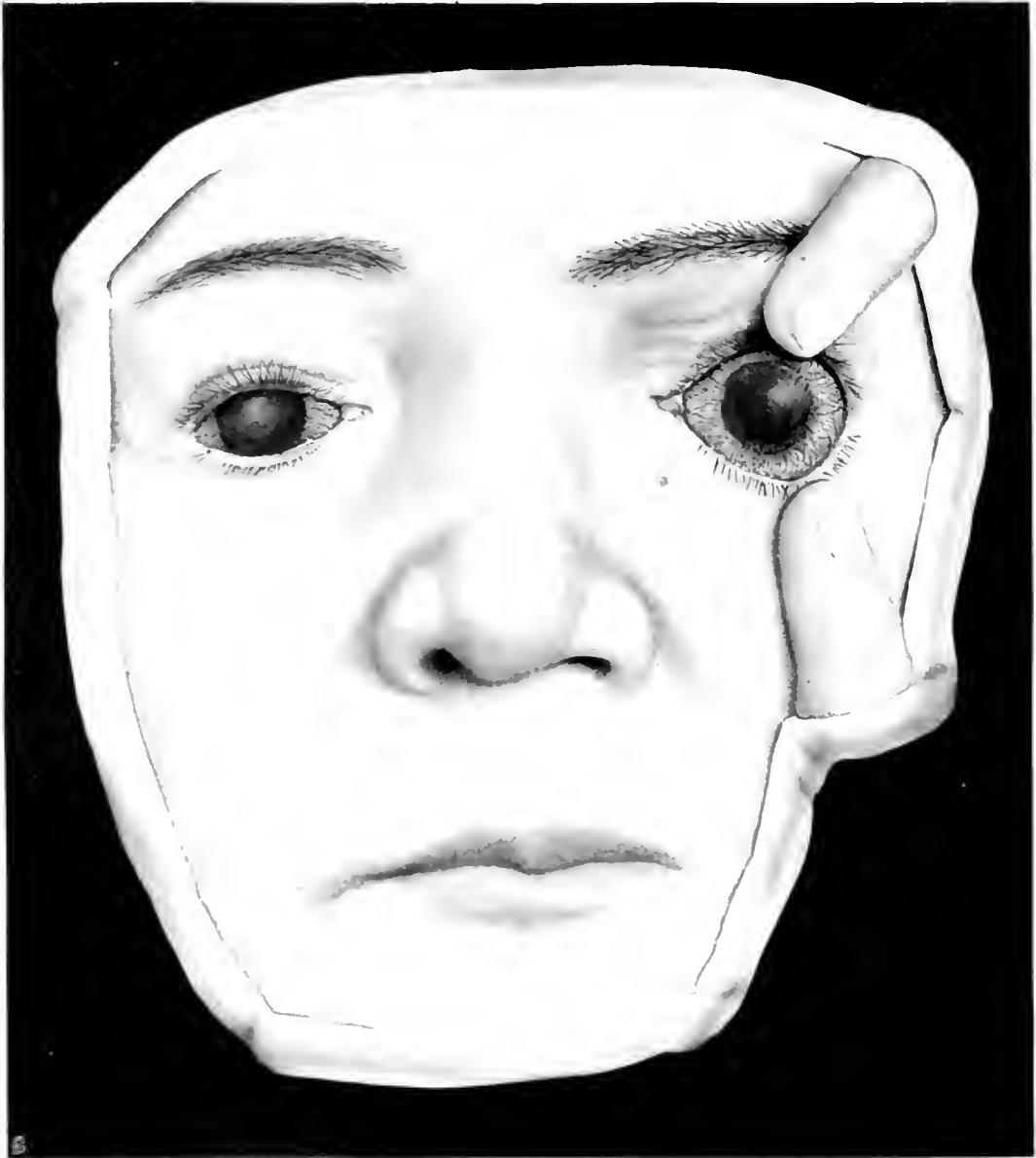


Fig. 48.
Keratitis interstitialis.

Interstitial Keratitis.

Plate XXXIII, FIG. 48; Plate XXXIV, FIGS. 49, 50; Plate XXXV, FIG. 51; Plate XXXVI, FIG. 52

Interstitial keratitis is only one of the names by which this affection is known. Perhaps the one most commonly used is parenchymatous keratitis, but it is not the best, because all diseases of the cornea invade its parenchyma more or less. Another name is syphilitic keratitis, which refers to its usual etiology, but it is not applicable to all cases, and, moreover, the condition is not one which is characteristic of syphilis in general, but rather of the hereditary variety. Deep keratitis, or keratitis profunda, is a correct name in that the pathological process extends deeply into the tissue of the cornea, but ulcers likewise penetrate to its deepest layers. The best names are interstitial keratitis, and diffuse keratitis, because the diffuse, uniform extension of the lesion throughout the entire cornea is in fact characteristic. The term uveitis anterior also seems to be justified, because the entire anterior portion of the uvea is apt to be involved.

Interstitial keratitis has pronounced **characteristics**, and usually starts and runs its course in a perfectly typical manner. It is always a symptom of some grave constitutional disease; therefore its onset informs the physician that it is his duty to make a thorough and accurate examination of the patient. While it may be the first symptom to attract attention, other symptoms can be found almost always, and from the combination of these the hitherto latent disease may be recognized. Hereditary syphilis is the fundamental cause in the great majority of cases.

The affection begins in one eye, sometimes simultaneously in both, with a delicate pericorneal injection, which soon becomes more intense and indicates a grave, deep-seated disease of the eye. Soon afterwards a triangular, cloudy, lusterless place may be seen with its base at the periphery of the cornea, its apex advancing toward the center. Other similar opacities then appear at other parts of the periphery, blend, and finally involve the entire cornea uniformly, giving it the appearance of glass that has been breathed upon, or

has been smeared with fat, and allowing little or nothing to be seen of the subjacent parts.

The opacity looks uniformly gray to the unaided eye, but with a magnifying glass and oblique illumination it can be seen to consist of numerous little, whitish spots. No gross lesions can be seen on the surface of the cornea, neither elevations like blebs, nor depressions, like ulcers, or defects in the epithelium, but with a magnifying glass the epithelium may be seen to be covered uniformly by many minute elevations, which give it a granular appearance. When Placido's disk is mirrored on such a cornea the concentric circles are nowhere much distorted, though their margins are indistinct.

As the opacity advances an extensive vascular development starts to accompany it. The newly formed vessels from the marginal network halt at the limbus, or advance only a little beyond it, and there form a reddish swelling about the cornea. The episcleral vessels, on the contrary, penetrate the deeper layers of the cornea, always being a little behind the opacity in its advance. In mild cases these vessels are few in number, and are often embedded in the opacity so that they can hardly be seen, but in severe cases they proliferate abundantly and may finally give the entire cornea the dirty reddish color of raw meat. These vessels are quite characteristic in appearance; they do not interlace, as in trachomatous pannus, but follow straight courses toward the center of the cornea, nearly parallel with one another, even when they divide into branches.

The interstitial keratitis is accompanied by a more or less marked involvement of the uveal tract, that varies from a simple hyperaemia of the iris to a very severe iridochorioiditis. An iritis is almost always present. It is indicated by the pericorneal injection, the swollen, discolored iris, and the tendency of the pupil to contract. In bad cases permanent posterior synechiae, and, perhaps, an occlusion of the pupil, will be left if the pupil is not dilated artificially during this stage, and the pupil often displays a constant tendency to contract, even when the instillations of atropine are frequent.

If an opportunity is presented to examine the eye with the ophthalmoscope at the commencement of the trouble, while the cornea is still transparent, large black spots may be perceived that indicate the presence of a chorioiditis, or of a uveitis anterior. Probably this affection is one of the commonest accompaniments of an interstitial keratitis, although the opacity of the cornea soon renders its detection with the ophthalmoscope impossible.

The intraocular tension is lowered a little in some cases. An

increase in tension is rare, and occurs only after the affection has lasted a long time, [but glaucoma sometimes supervenes].

The vision is always greatly impaired during the attack. It is seldom that even the largest type can be read, and as a rule the patients can see only movements of the hand. Very often both the test of vision and the examination of the eye are rendered difficult by an intense photophobia and an accompanying profuse lachrymation.

This photophobia is the principal subjective trouble of which the patients complain. Pain is absent at first, but comes on later, though it is rarely very severe when proper treatment has been instituted. [The pain, photophobia, and lachrymation are generally proportionate to the degree of vascular development.]

Quite a number of **variations** of this clinical picture, with its complications, are met with. [Thus in some cases only one triangular segment of the cornea is affected, and in a good many the opacity appears first in the central part of the cornea and spreads toward the periphery. In some of the latter the center becomes far more opaque than the peripheral zone, while in others an opaque circle is formed about the more or less transparent center, described by *Vossius* as annular keratitis.] In spite of these variations, many of which depend on the severity of the attack, sufficient of the characteristic symptoms are present, as a rule, to render the diagnosis not difficult. Superficial keratitis is excluded by the absence of ulcers and of gross prominences on the surface of the cornea, so that the circles of Placido's disk reflected on it are not distorted, although their margins are not sharply defined. The general bodily condition is also of aid in the differentiation. Superficial keratitis is generally a symptom of scrofulosis, and is accompanied by diseases of the nose, eczema and enlarged glands [that are soft and apt to undergo caseation and to break down;] interstitial keratitis is usually a symptom of hereditary syphilis, [and then the lymphatic glands are small, hard, painless, and show no tendency to break down. Suppuration has been known to take place in the parenchyma of the cornea in a few cases.]

Interstitial keratitis is almost invariably bilateral, sometimes attacking both eyes simultaneously, but usually first one eye and then the other after a short interval, more rarely after an interval of several weeks or months. It is generally met with in persons between the ages of 6 and 18, exceptionally in persons over 20, when the affection is apt to run a milder and atypical course, and, in rare

instances, may attack only one eye. As a rule the patients are pale, unhealthy, ill-nourished children.

Interstitial keratitis is never a local disease, [even when it follows a traumatism.] Formerly it was confounded with lymphatic or serofulous keratitis, and *Hutchinson* was the first to learn that it is usually due to hereditary syphilis. He called attention at the same time to another symptom which is often associated with it, and is considered to be a sure sign of hereditary syphilis, the so-called **Hutchinson teeth** (see Plate XXXIV, Fig. 49).

By **Hutchinson teeth** is understood a deformation of both upper middle incisors of the second dentition, such that their sides converge downward, and each lower margin presents a crescentic notch, instead of a sharp cutting edge. This notch is commonly demonstrable until the patients are about 25 years old, but after that age the corners are generally worn off, or crumbled away. Other dental anomalies that often accompany this typical deformation are an abnormal smallness of single teeth, particularly of the canines, and an irregular arrangement and formation of the teeth, with wide spaces between them.

These teeth must not be confounded with **rhachitic teeth**, which have horizontal furrows, due to defective enamel (see Plate XXXIV, Fig. 50).

A third symptom that appears very often together with an interstitial keratitis and Hutchinson teeth, is a deafness that cannot be ascribed to any objective clinical lesion in the ears. These three symptoms form the so-called **Hutchinson triad**, which is accepted as positive proof of the presence of hereditary syphilis. I have never seen a patient with the Hutchinson triad in whom other symptoms could not be detected.

Arlt and *Foerster* called attention to an affection of the knee joint as often preceding or following an attack of interstitial keratitis, and more heed has been paid of late to the frequent occurrence of **joint diseases** in hereditary syphilis. *Fournier* found them in 82 out of 212 cases. The joint most apt to be affected is the knee, then the elbow, more rarely others. The arthritis generally precedes the interstitial keratitis, though it may occur simultaneously, or, less often, may follow, and is frequently bilateral. The condition is almost always that of a serous effusion into the joint, which takes place with a moderate amount of rheumatic pain and little or no fever. *Fournier* believes that these joint affections depend on disease



Fig. 49. Hutchinson's Teeth.

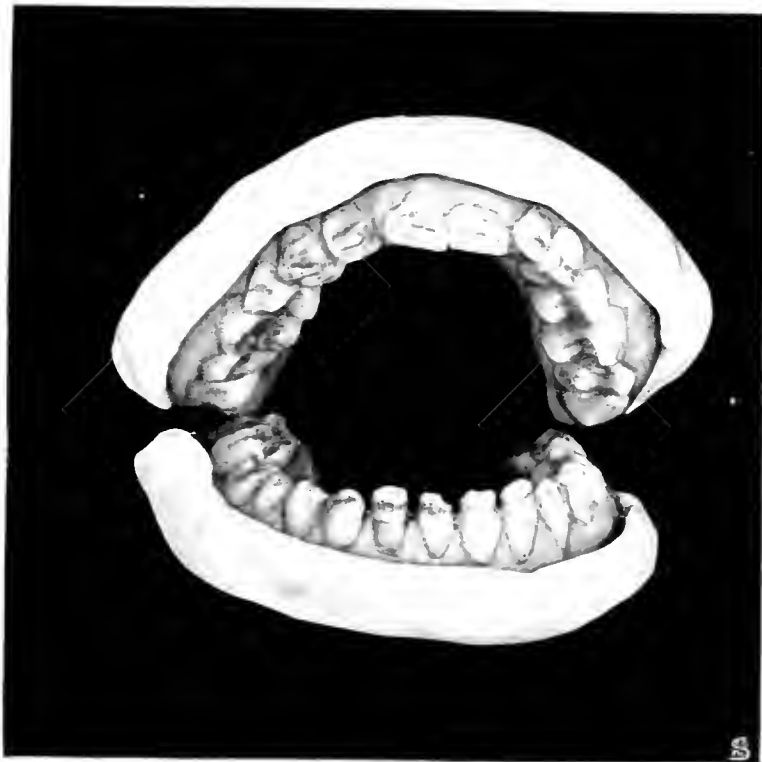


Fig. 50.
Rhachitic Teeth.

of the bones, but other syphilographers consider them to be due to a primary inflammation of the synovial membranes.

Other points that should be taken into account are the state of the lymphatic glands, the peculiar conformation of the face and skull (see Plate XXXVI, Fig. 52), the bridge of the nose, which is often sunken (see Plate XXXIII, Fig. 48), any ozaena, or any blennorrhoea of the lachrymal sac that may exist, and the presence of rhagades at the angle of the mouth or elsewhere on the face (see Plate XXXV, Fig. 51).

The history of an antecedent infection of the father or mother with syphilis, of miscarriages caused by dead foetuses, and of the deaths of many of the older children in infancy, is important when taken in connection with the objective findings.

Diagnosis

Often the diagnosis can be made very easily; often, on the contrary, it can be made only after a most rigid and careful investigation, and there are cases in which hereditary syphilis can be excluded, and the keratitis must depend upon some other disease, such as scrofulosis, chlorosis, or, above all, tuberculosis. Among the diseases that rarely act as the cause may be mentioned acute articular rheumatism, malaria and influenza.

Course

The course of the disease is very slow; three to twelve months often elapse before the inflammatory symptoms abate and the opacity clears up. The physician will find it to be his best policy to state this at once, to recommend patience, and, when only one eye is affected, to foretell that the other will soon be attacked, lest fright be occasioned and the treatment blamed when this occurs.

Nature

[The nature of interstitial keratitis is still uncertain. It is the opinion of some writers that when it appears as a symptom of hereditary syphilis it is produced by the direct action of spirochaetae upon the tissue of the cornea. Spirochaetae have been found in the corneas of syphilitic foetuses and infants, but usually they had produced no visible reaction, and they are not easily demonstrable in the corneas of older children suffering from interstitial keratitis. Other writers think it to be an indirect manifestation of the disease. *Derby and Walker* (Trans. of the American Ophthalmological

Society, 1913) suggest that it may be an anaphylactic phenomenon. It may be that the spirochaetae which wander into the cornea of a syphilitic child, or the syphilitic toxine, sensitize the tissue so that when the latent virus again becomes active at a later period the increased quantity of syphilitic material in the circulation causes the tissue to react with the production of an interstitial keratitis. The slight influence exerted by antisymphilitic treatment in most cases would seem to uphold the view that the corneal affection is not produced in them directly by the spirochaetae, while the prompt response to the same remedies when the keratitis appears as a symptom of acquired syphilis suggests that in these cases the lesion may be due to the direct action of these organisms.]

Prognosis

The prognosis is fairly good, aside from the fact that no medical treatment is able to arrest or shorten the course of the disease. The corneal opacities are accustomed to pass away, even after the affection has lasted a long time. The process of clearing up commonly begins at the margin of the cornea, and the opacity in the center is the last to disappear. Usually the eye regains good, or at least useful, vision; less commonly dense opacities are left permanently. [In some cases the cornea seems to become softened by the disease, and to bulge forward, thus creating a myopia, and it has happened in quite exceptional cases, which were not properly treated, that the uveitis resulted in a phthisis bulbi. When a dense central opacity is left an optical iridectomy, with or without tattooing, should not be done until a year at least has elapsed.]

Treatment

Treatment must be both local and general. Moist, warm compresses alleviate the symptoms of irritation and inflammation in the early stages. The eye may be bathed frequently with hot water, or a large pad of cotton, wet with a warm 2 to 4% solution of boric acid may be placed over the eye, covered with rubber tissue, and secured in place by a bandage. The latter gives particularly good service at night; when worn during the day it should be changed once or twice. Chamomile tea is a favorite household remedy, but its use should be discouraged, as it is not cleanly, and it sometimes floats particles of dust into the eye.

When the photophobia is great the eyes should be protected from



Fig. 51.
Rhagades on face in hereditary Lues.

the light by keeping the room moderately darkened, and later by having the patient wear a screen, or protective glasses.

Complications on the part of the iris are almost invariably present, and the most important measure to be taken as soon as they appear is to instill a 1% solution of atropine sulphate several times a day until the pupil is dilated, and then to maintain the dilatation. Neglect of this precaution may result in the formation of posterior synechiae, perhaps of occlusion of the pupil, and these will permanently impair the vision. [The local use of atropine is necessarily prolonged in these cases, but it should not be maintained without careful oversight. We know that glaucoma sometimes occurs, and *Agnew* observed that the circulation at the root of the iris is apt to be impeded as a result of a prolonged use of atropine, so he was accustomed to occasionally alternate the atropine with a meiotic for the benefit of the circulation. Hence it is certainly advisable to watch the tension carefully throughout the course of the disease, particularly in its later stages, and to intermit the atropine from time to time, when the symptoms of uveitis are not too urgent.]

Formerly an operation known as ***peritomy of the cornea*** was performed in some cases in which the vascular development was great and long continued, by cutting the vessels with a knife, or by excising a strip 1 or 2 mm. wide down to the sclera around the entire circumference of the cornea. But experience has proved that the vessels reunite after a while, and that this is followed by an attack of inflammation more violent than the preceding, so the operation now has been generally abandoned.

After the inflammation has subsided an attempt may be made to hasten the clearing up of the opacities by the insufflation of powdered calomel, or by massage with the yellow oxide ointment. We are accustomed at the University Clinic in Berlin to use for this purpose a weaker ointment than the one originally prescribed by *Pagenstecher*.

[Dionin may also be used, as well as subconjunctival injections of saline. About a dozen of these injections may be made at intervals of three days, but it is not wise to continue them if they are not well borne, or if they do not seem to be productive of good results. Some prefer subconjunctival injections of mercury, although they are more painful and it is hardly certain that they are more efficacious than those of saline. The solutions employed are usually made with the cyanide of mercury, about 1 to 5000. *Darier* states that the preparation which is least painful and best borne is a 1 to 500 solution of mercury salicylarsenate.]

General treatment must be guided by the general condition, [and the fundamental disease.] Weakly patients should be given a strengthening diet. Favorite remedies are those that contain iodine, such as iodide of iron, cod liver oil with iodine, and mineral waters holding iodine in solution. In the later stage of the affection a sojourn in the country, where the air is good, is to be highly recommended. [Saline baths are sometimes useful.]

The fundamental cause in the large majority of cases is hereditary syphilis, and this calls for its ordinary treatment with mercury, iodide of potassium, [and salvarsan. The best way to give mercury is by the intramuscular, or intravenous, injection of the cyanide, or by the intramuscular injection of the salicylate, or of the bichloride. The next best way is by inunction either with the ordinary blue ointment, or the combination of belladonna with mercury said to have been suggested by *Arlt*. When neither of these ways can be employed suitable doses of the biniodide or bichloride may be given by mouth. In the opinion of many observers the antisiphilitic remedies have little influence on the interstitial keratitis, while others think that they shorten its average duration. Many of the latter give several intravenous injections of salvarsan at intervals of a week or more, under the guidance of the Wassermann reaction, administering mercury and iodide of potassium at the same time. It should always be borne in mind that, even though the keratitis may not respond quickly, antisiphilitic treatment should be carried out to save the patient from other, and perhaps more permanently serious lesions, and that it should be maintained a long time after the ocular trouble has disappeared.

Harlan has seen several cases in which an interstitial keratitis cleared up with remarkable rapidity when the children suffering from it had an intercurrent attack of measles.]

When tuberculosis is the cause the treatment appropriate to that disease is indicated. General nutrition is to be cared for first of all. [Fresh air, nutritious food, and hygienic surroundings are our main reliance. In addition tuberculin treatment may be tried.] Internally we may give creosote, or its active principle guaiacol, which tastes less unpleasantly and is better borne. A 1 or 2% solution of guaiacol may also be used for subconjunctival injection, which is also said to be able to control a too violent ocular reaction provoked by tuberculin.

In the rare cases where interstitial keratitis is caused by malaria, rheumatism, or influenza, the general treatment appropriate to these diseases is likewise indicated.

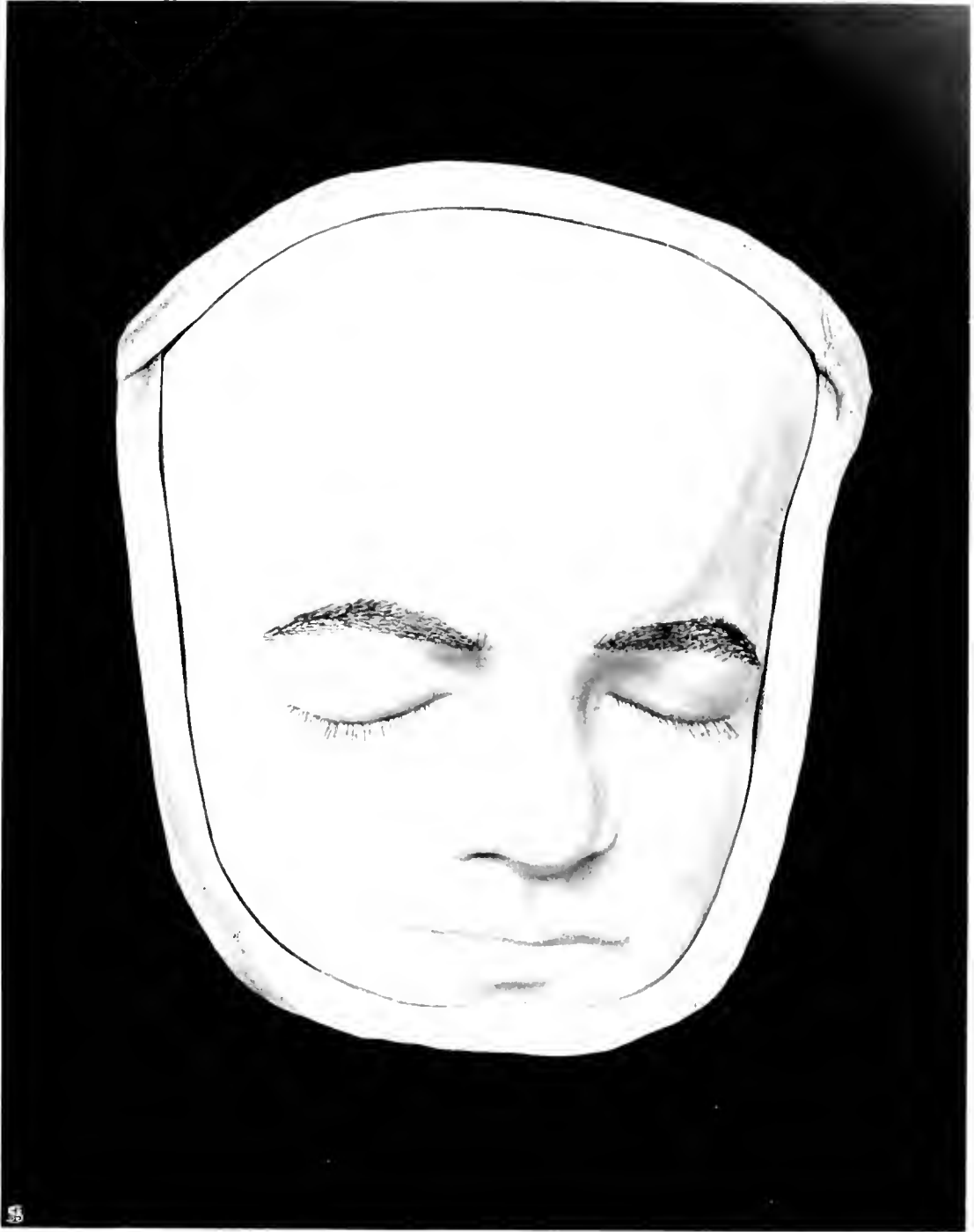


Fig. 52.
Head formation in hereditary Ties

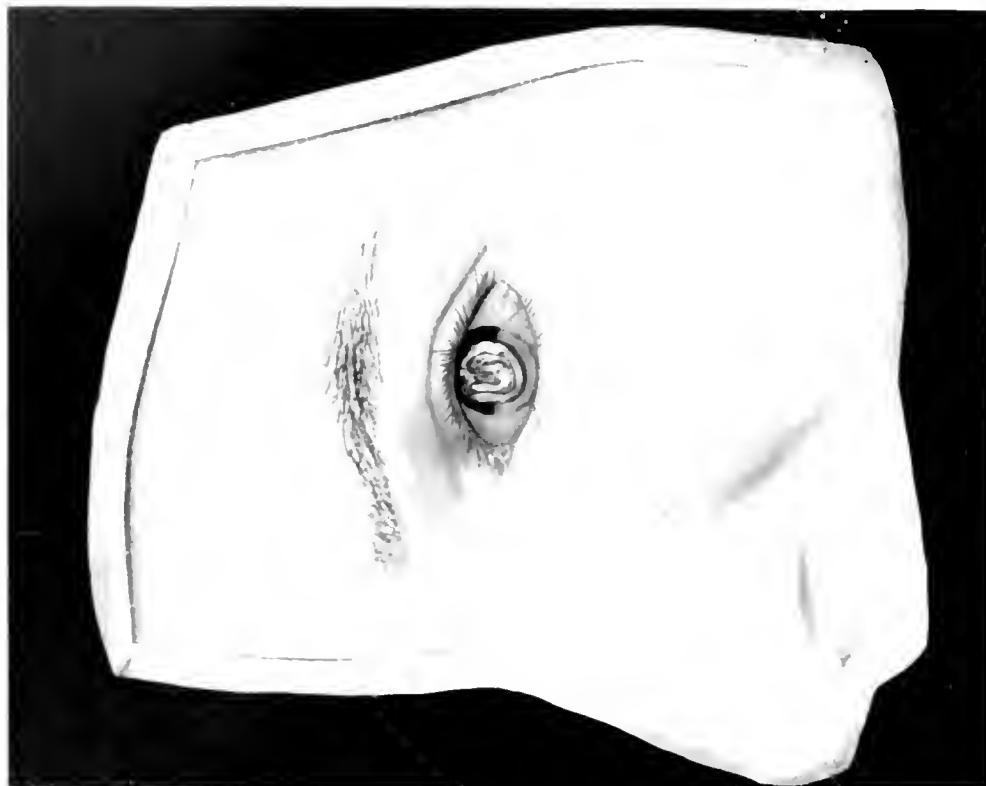


Fig. 51.
Advanced *Ulcus serpens*.



Fig. 53.
Ulcus serpens with Hypopyon.

Serpiginous Ulcer of the Cornea

Plate XXXVII, Figs. 53, 54

The normal epithelium of the cornea is so dense that the ordinary pus agents are unable to penetrate it, and pus containing staphylococci, streptococci, and even the virulent pneumococci usually found in that coming from a suppuration of the lachrymal sac, may flood the eye continuously for a long time without producing any considerable inflammation, or at least without making any impression upon the intact cornea. Gonococcal pus alone is able to corrode the intact corneal epithelium when it stagnates and induces maceration. All of the other pus agents must find an artificial place of entrance if they are to penetrate into the cornea. Such an entrance is usually furnished by a wound, which may be nothing more than an insignificant, superficial abrasion. If it happens that at the time this wound is produced pus agents are present in the conjunctival sac, on the foreign body, or in secretion coming from the lachrymal sac, these agents may enter the wound, develop in a short time luxuriant colonies beneath the epithelium, which is then cast off, and start what is known in the modern sense as a **septic ulcer** of the cornea. A characteristic feature of such an ulcer is a collection of pus in the anterior chamber, which appears before long, is called a hypopyon, and gave the disease its old name, hypopyon-keratitis. The name **serpiginous ulcer** is more appropriate, because it is indicative of its great tendency to burrow rapidly and continuously beneath the epithelium.

Course

[The inflammation sets in with severe pain in the eye and head which is worse at night. In the earliest stage of the ulcer a cloudiness appears about the defect in the epithelium together with one or more yellowish spots, usually at its margin. After the epithelium has been undermined and cast off the ulcer appears grayish, with its margins more opaque than its center. Polymorphonuclear leucocytes collect just beyond the margin to form a yellowish infiltration, which is apt

to be greatest on one side, where the pneumococci are most numerous and the progression is most rapid. As a rule the ulcer starts in or near the center of the cornea, because this is the part most exposed to injury.]

Iritis sets in regularly as soon as the ulcer has attained a certain degree of development. [It is due to the diffusion of the toxic products of the infecting organisms into the aqueous, which is likewise responsible for the hypopyon. The latter is composed of sterile pus, at least until perforation has taken place, because Descemet's membrane is very resistant to the passage of cells and of micro-organisms.]

The appearance of a serpiginous ulcer is shown very well on Plate XXXVII. In Fig. 53 we see the disk-shaped ulcer with its yellow, elevated margins. The cornea in its vicinity appears misty and oedematous. A crescentic, narrow hypopyon occupies the lower part of the anterior chamber. A just beginning pericorneal injection indicates the onset of an iritis.

Fig. 54 shows the condition after the ulcer has destroyed the greater part of the surface of the cornea. More than one-half of the anterior chamber is filled by the hypopyon. A slight swelling of the lids and a very marked pericorneal injection bear witness to the presence of a severe iritis, of which nothing can be seen directly.

Prognosis

This is a common and serious disease which, if left alone, often results within a few days in incurable blindness of the eye. After eating away the greater part of the surface of the cornea the ulcer invades the deeper tissues, causes perforation at some place, and then generally ceases to progress, [although in some cases the infection may spread into the eye and cause panophthalmitis.] The loss of substance is then replaced by cicatricial tissue. [The vision is apt to be very seriously impaired, even with the best and most energetic treatment, as an opacity of some density is usually left in the central part of the cornea in the mildest and most benign cases.

Treatment

The first thing to be done in the line of treatment is to determine the bacteriological cause of the ulcer. After the eye has been cocaine-ized a bit of material should be taken from the margin of the ulcer where the infiltration is greatest, placed on a slide, stained, and examined under the microscope, because very similar clinical pic-

tures are produced by pneumococci and diplobacilli, and yet the two conditions are not amenable to the same treatment. Indeed the treatment curative of the one may be distinctly harmful to the other.

When **pneumococci** are the active agents] we will find the source of infection very often in a disease of the lachrymal sac. It is then imperative to either open the lachrymal sac by a free incision through the skin, and to pack it with iodoform gauze, or to extirpate it. [The latter operation is preferable, both because it leaves a less noticeable scar, and because the immediate and total removal of the constant source of infection is urgently indicated. We can hardly expect to accomplish much in the way of disinfection of the ulcer as long as it continues to be inundated with pus containing virulent pneumococci, and, as this comes from all parts of the inflamed mucous membrane of the lachrymal sac, the presence of any portion of this membrane remains a menace to the eye. For this reason extirpation is also preferable to dacryorhinostomy in these cases. *Wessely* has recently advocated the injection of tincture of iodine into the lachrymal sac, in place of extirpation, and claims that in 80% of his cases the secretion ceased after from one to four injections, but this observation needs to be confirmed before the procedure can be recommended. The radical cure of every case of dacryocystitis is urged by many writers as a prophylactic measure against serpiginous ulcer.] No good whatever is accomplished by probing the lachrymal passages.

Recently the subcutaneous injection of pneumococcus serum has been used with success. [*Roemer* recommends doses of from 25 to 50 ccm., and says that smaller ones are ineffective. A strong local reaction takes place at the margin of the ulcer for a couple of days, and then the progressive edge melts away. A transient increase of the hypopyon may also occur without indicating any progression of the disease. When the progressive margin does not begin to melt away at the end of 24 to 48 hours, especially when signs of progress are present, recourse must be had to cauterization. When the signs are those of improvement the injection should be repeated on the second or third day, and thereafter as conditions indicate.]

Locally some drops of fresh, undiluted chlorine water may be poured over the surface of the ulcer every two hours, [or, if the serum treatment has been adopted, serum may be dropped on the ulcer several times a day, in order to bring the antibodies in more direct contact with the diseased tissue, but such a local use of the serum alone can accomplish very little]. If the margins of the ulcer

are progressive they should be touched with the galvanic cautery [after the eye has been anaesthetized, whether the serum treatment is used or not. It is well to stain the ulcer first with fluorescein, to reveal its extent with exactitude, and *Darier* recommends powdering it with dionin in order to set up an intensive detersion of the morbid tissue, and to render the parts as analgesic as possible. Although as a general rule the cautery should be applied as lightly as possible to the cornea, in order to avoid the creation of dense opacities, the margin of a rapidly spreading pneumococcal ulcer should be cauterized deeply, to arrest the process and to save the transparency of the rest of the cornea. The burned tissue may act as a foreign body, so it is best to remove it as soon as the cauterization has been performed.

The use of steam instead of the galvanic cautery has been recommended of late several times. *Wessely* states that in his experience a single application of hot steam sufficed to arrest the disease in 75% of the cases, and that the final average vision was much better than that usually obtained, because the cicatrices were more delicate and less uneven.]

Iodoform, or better airol, may then be dusted into the conjunctival sac, where it forms a paste that soon hardens into a firm crust over the base of the ulcer.

[Atropine should be instilled regularly from the onset of the first sign of iritis, and the pupil kept dilated if possible. If the pupil contracts in spite of this, the danger of the formation of posterior synechiae is very grave, but may be warded off sometimes by subconjunctival injections of saline, or of cyanide of mercury.]

When the ulcer is far advanced *Saemisch's* operation may be performed, [but it is better to introduce a keratome at the lower margin of the cornea, allow the aqueous to escape slowly, and then to remove what we can of the hypopyon without undue violence. Frequently it is wise to do this immediately after the ulcer has been cauterized. Atropine should then be instilled, the eye bandaged, and the patient put to bed.

After the progress of the infection has been checked and the ulcer has become clean, the process of repair may be aided by the use of hot applications. In some cases at least the opacity left by the cicatrization seems to be capable of becoming more transparent under massage with the yellow oxide ointment, if maintained for weeks or months. When a dense white scar is left in the center of the cornea

the vision may sometimes be improved by an optical iridectomy, combined with tattooing of the cicatrix.

*When the ulcer is due not to the pneumococcus, but to the diplo-bacillus, **cauterization must never be performed**, as it is apt to render the condition worse. The only treatment needed is an instillation of a 5% solution of zinc sulphate every hour or two, with a daily application to the ulcer of a 20% solution of the same drug.]*

Leucoma of the Cornea Caused by Small Pox

Plate XXXVIII, FIG. 55

The only portion of the cornea that is transparent when regenerated is the epithelium. When any other part is destroyed a scar is left which varies in density and thickness with the depth to which the normal tissue has been replaced by cicatricial. A delicate, superficial, but visibly gray cicatrix is called a **nubecula**; one that is more dense, a **macula**; one that is totally opaque and white, a **leucoma** of the cornea. A leucoma may be partial, like the one pictured in Fig. 35 on Plate XXIV, or total, like the one shown in Fig. 55.

The black line in the cicatrix in Fig. 35 shows where a part of the iris was caught, as happens in many cases. Such a condition is known as a **leucoma adhaerens**, the adhesion of the iris to the posterior surface of the cornea as an **anterior synechia**. Its existence is a proof that a perforation of the cornea occurred at this place.

Treatment

Nothing can be hoped for from treatment, or from time, so far as the removal of a dense **leucoma** is concerned. If it is total, vision is reduced to perception of light, but if it is partial, an artificial pupil may be made where the cornea is still transparent (see Fig. 35). The disfigurement occasioned by the leucoma may be lessened by **tattooing** it black with Chinese or India ink. [This operation should never be performed as long as any pathological process is present in the eye, and is not advisable when the cornea is thin and staphylomatous, or when the iris has prolapsed through the perforation of the cornea, as in Fig. 35, because there is then considerable danger of setting up a severe inflammation, and cases have been reported in which an attack of sympathetic ophthalmia destroyed the other eye. The best results seem to be obtained when the cornea is of normal or greater than normal thickness, and the iris, if involved, is simply attached to its posterior surface. The usual way of perform-

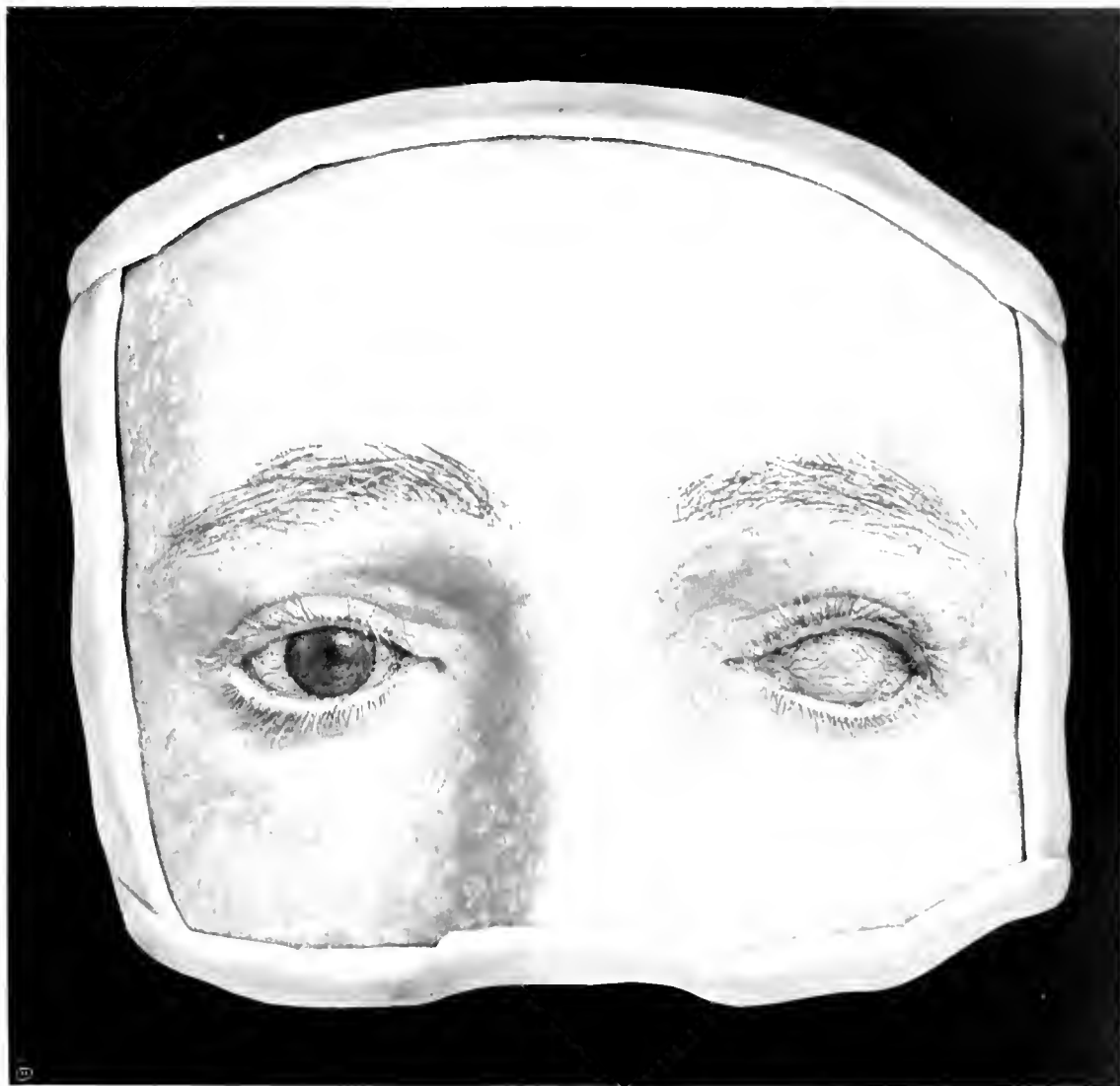


Fig. 55
Leucoma corneae totale.

ing the operation is to prick the superficial layers of the cicatrix repeatedly with a tattooing needle, held obliquely, until the entire area that is to be stained is roughened by the punctures. A sterile paste of India ink is then rubbed into the surface with a spatula. The eye is then washed off with sterile water or saline, the effect noted, and the operation repeated until the area remains black. Atropine should then be instilled and the eye bandaged for a few days until the reaction is past.

Barck suggested that the cosmetic appearance could be improved still more if a round, central area was tattooed to represent the pupil, the periphery to resemble somewhat the iris, and the two separated by a narrow ring of unstained tissue to show the pupil by contrast. To do this he devised an instrument with two concentric circular knives, the smaller one to demark the pupil, the larger to show the inner limit of the iris. The tattooing was then done as before. Local anaesthesia alone is necessary. It is best to avoid the use of a fixation forceps, if possible, as any wound made in the conjunctiva is apt to become stained. The duration of the stain in the cornea cannot be foretold; sometimes it lasts for many years, sometimes it disappears in a few months.]

Nubeculae and **maculae** frequently clear up a great deal in time, and this process may be accelerated by stimulants that increase the metabolism, like powdered calomel, yellow precipitate ointment, 2 to 5%, and douches of hot steam, but this can be done only in fresh cases. *Dionin* has been recommended much of late, used at first in a 5 or 10% solution, later in the form of powder, [but it is a question whether its value has not been overestimated; it is certainly not reasonable to expect too much of it in old leucomas.]

Sometimes both of the corneas of an infant appear milky and resemble the sclera. Such a condition was formerly thought to be due to a fault of development, but this is not the case as a rule; it is usually due to a **keratitis interstitialis in utero** caused by hereditary syphilis. Antisyphilitic treatment is therefore indicated.

The **arcus senilis**, or gerontoxon of the cornea, is a non-inflammatory physiological opacity of the cornea which develops in old age, sometimes early, sometimes late. It consists of a narrow, gray line that runs near and concentrically with the margin of the cornea. It begins almost always as an arc at the upper margin, soon followed by a similar arc at the lower margin; the ends of these two arcs

extend until they meet along the inner and outer sides of the cornea and form a complete ring. Its outer edge is sharply defined and is always separated from the limbus by a band of clear cornea (see Plate L, Fig. 74).

This senile are is not a progressive opacity, but as soon as it has formed a complete circular band, 1 or 2 mm. broad at most, it remains stationary.

Another of the non-inflammatory opacities of the cornea is the **band-shaped**, or zonular (see Plate XXV, Fig. 36). This forms a white stripe, 2 to 4 mm. broad, horizontally across the cornea just below its center. Each of its ends is separated from the margin of the cornea by a narrow, transparent zone. The course of its development is slow, requiring years. It consists of a deposit of lime salts consequent upon a disturbance in the nutrition of the cornea.

There is no treatment for this condition, which is really of little practical importance, as it is generally seen in eyes that have already been rendered blind by an insidious uveitis, [sympathetic ophthalmia, or glaucoma.

Very rarely this condition may be congenital, and it should be remembered that it may develop in eyes that were previously normal. Either this condition, or one like it, was observed by *Fuchs* in a physician who had been accustomed to insufflate calomel into his eyes for a long time, and has been seen to develop in the eyes of workers in naphthalin and aniline dyes.]

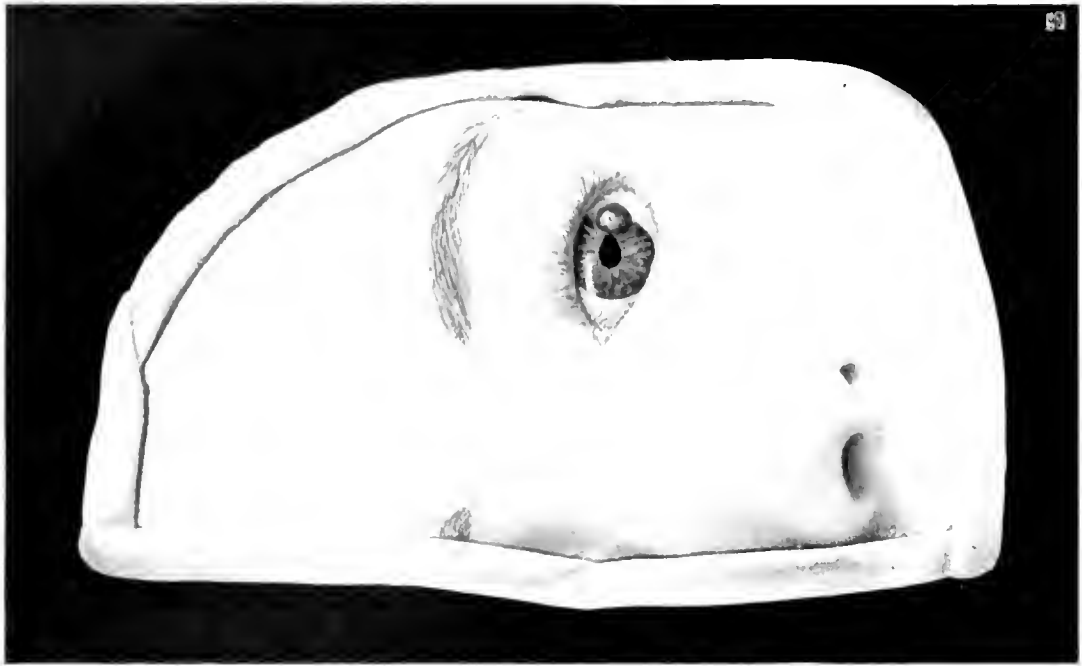


Fig. 57.
Staphyloma corneae parziale.

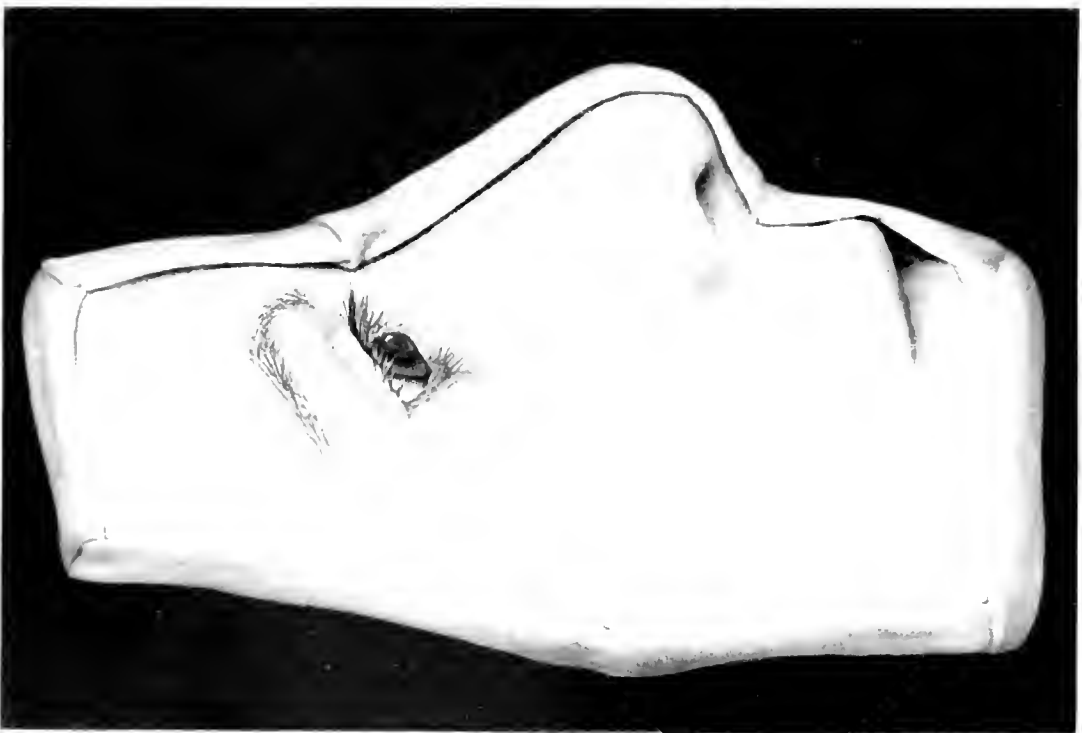


Fig. 56.
Keratoconus.

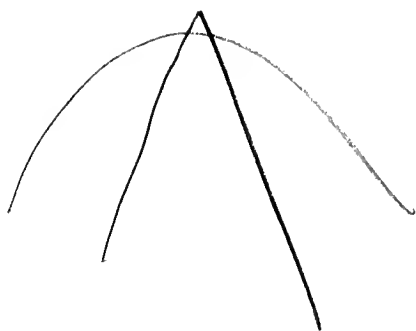




Fig. 58.
Buphthalmus. Cornea globosa



Fig. 59.
Staphyloma orneae totale.

Ectasias of the Cornea.

KERATOCONUS: *Plate XXXIX*, FIG. 56

STAPHYLOMA CORNEAE PARTIALE: *Plate XXXIX*, FIG. 57

BUPHTHALMUS; CORNEA GLOBOSA; KERATOGLOBUS:
Plate XL, FIG. 58

STAPHYLOMA CORNEAE TOTALE: *Plate XLI*, FIG. 59

Staphyloma of the cornea is a protrusion of a cicatrix formed chiefly of prolapsed iris, at a place where the cornea has been destroyed, and caused to bulge forward by the intraocular pressure. The way in which it is produced, as the final result of a suppuration of the cornea, is illustrated in the left eye shown in Fig. 38 on Plate XXVI, where a prolapse of the entire iris can be seen to have taken place after destruction of the cornea, and to be already beginning to be protuberant. A staphyloma may be partial (see Fig. 57), or total (see Fig. 59).

At first, and in small ones, the black pigment of the iris predominates, but later this tissue becomes so cicatricial and so stretched that the staphyloma appears as a gray white protrusion traversed by broad blood vessels.

Treatment

A partial staphyloma of the cornea should be abscised, as the defect left usually becomes closed by a cicatrix composed of firmer connective tissue than can be produced by the thin iris.

After abscission of a total staphyloma it is best to suture the margins of the large opening together, so as to leave simply a stump.

[A staphyloma of the cornea is a serious condition, not only on account of the disfigurement it occasions, and of the irritation it produces and to which it is subject when it becomes too large to be covered perfectly by the lids, but also because it favors the development of glaucoma, even when small, by displacing the sinus of the anterior chamber. Therefore, as soon as a corneal cicatrix shows a tendency to bulge, an attempt should be made to prevent the forma-

tion of a staphyloma by performing an **iridectomy**, provided of course that any portion of the anterior chamber remains. Such an iridectomy should be made, if possible, where it may serve for an artificial pupil.

When the staphyloma progresses in spite of an iridectomy, or when it had formed before the case came under observation, it should be **abscised**, and the defect covered immediately with a flap taken from the bulbar conjunctiva. First we form the flap by incising the conjunctiva parallel and close to the margin of the cornea half way or more around its circumference, with the mid-point of the incision nearest to the staphyloma, and by dissecting it free from the sclera until it can be drawn easily over and beyond the proposed wound in the cornea. Two sutures are then inserted, one at each point where the flap crosses the limbus, so that they will secure the flap in place, but not tied. The flap is then pushed back, the staphyloma abscised, the flap drawn smoothly over the defect, the sutures tied, both eyes bandaged and the patient put to bed. The patient may have the use of his good eye after three or four days, but the other should be dressed and rebandaged daily for about two weeks. Healing takes place rapidly, with the formation of a firm, resistant scar. The portion of the flap that lies on the normal cornea does not adhere, but soon atrophies. An optical iridectomy may be performed some weeks later, if it has not already been made.

We should remember that this operation may rekindle the old iridocyclitis that accompanied the primary, ulcerative cause of this condition, and that sympathetic ophthalmia may supervene. This danger is not great enough to deter us from trying to save an eye with some degree of more or less useful vision, but the case is different when we have to deal with a total staphyloma. Left alone the best possible vision in this condition is perception of light, and this is lost when we operate. The deformity, irritation, and danger of glaucoma, or of drying and ulceration of the surface of the protrusion, demand intervention, and the question in any given case is whether we should enucleate, eviscerate, or reduce the eye to a stump in order to form a better support for an artificial eye. This the surgeon must decide from the other conditions present.

Critchett's operation of abscission is perhaps the best known, but in this the needles are passed through the anterior part of the eye, wound the ciliary body and increase the danger of sympathetic ophthalmia.

For this reason *Knapp's* operation is preferable. Two sutures

are inserted in the following manner, one to the nasal, the other to the temporal side of the vertical meridian of the cornea: A curved needle carrying a suture is passed horizontally through the conjunctiva and the outer layers of the sclera about 4 mm. above the margin of the cornea, and then passed in the reverse direction horizontally through the conjunctiva and scleral layers at the same distance below the cornea. The vertical loops of thread thus placed are drawn aside, a Graefe knife is passed horizontally through the base of the staphyloma and a section made downward; the lower part of the staphyloma thus cut away is seized with forceps and the abscission completed with scissors. The sutures are then drawn tight and tied.]

Keratoconus is a rare condition that generally affects both eyes. The central portion of the cornea protrudes like a cone, very gradually, with no inflammatory symptoms, and remains transparent for a long time, until the apex of the cone begins to cicatrize and to become opaque as the result of the enormous stretching it has undergone.

Etiology

The etiology of keratoconus is obscure. Some disturbance of the nutrition of the cornea causes its central portion to become weak and thin, so that it can no longer successfully resist the intraocular pressure, which drives it forward. [It may be congenital, but is more apt to appear about the age of puberty, and is more common in women than in men.

Perhaps it may be due to an embryological defect in the mesoblast, that forms the interstitial tissue of the cornea, as suggested by *Tweedy*; or perhaps *Sattler* is right in thinking it a developmental tissue aberration of the central regions of the cornea, which, under certain exciting conditions, generally during adolescence, advances, and may again advance after a long spontaneous arrest.

Diagnosis

When the cone is well marked it can be recognized without difficulty by viewing it in profile, but when it is slight we must rely on other means. The circles of Placido's disk, mirrored on the surface of such a cornea, are small at the center and elongated toward the periphery. The mires of the ophthalmometer appear much distorted, as the principal meridians of curvature are seldom at right angles.

The skiascope reveals a high degree of negative aberration, so much so sometimes that the bright central illumination moves in a direction opposite to the movement of the mirror, while the peripheral movement may be with the mirror. The reflex from the cornea is also seen to be triangular, with its base at the periphery. The details of the fundus are distorted when seen with the ophthalmoscope, in either the upright or the inverted image. A pulsation of the apex of the cone, synchronous with the pulse, can sometimes be seen, especially with the ophthalmometer. The apex of the cone becomes so thin that it readily dimples when touched with a probe, and is prone to be anaesthetic.

Prognosis

The prognosis is not good. The change is apt to increase until the vision is badly impaired, but it is usually arrested after a while, and a very few cases have been reported of spontaneous improvement. In other cases the process seems to have been checked for years and to have recommenced in later life. When the apex of the cone is very thin it may be ruptured by a slight traumatism, but spontaneous rupture is not known to have taken place.]

Treatment

Treatment is far from satisfactory. The patient should be given a strengthening diet, while eserine or pilocarpine may be instilled for a long time in the hope of reducing permanently the intraocular tension, but an iridectomy is better for this purpose. The apex of the cone may also be destroyed with the galvanic cautery in order to obtain a more resistant cicatrix.

[Cases should be divided for the purpose of treatment into those who can, and those who cannot be given useful vision without an operation, but no given case should be included in the latter class until the most painstaking efforts have failed to improve the vision. The **fitting of glasses** is peculiarly difficult, because the refractive error cannot usually be corrected accurately with spherical or cylindrical lenses. The refraction at the apex of the cone is considerably myopic, but the myopia decreases rapidly down its sides, and the peripheral parts of the pupil may be hypermetropic. *Raehlmann* introduced the use of hyperbolic glasses to correct the error, but they have not proved very satisfactory, though useful in some cases. *Lohnstein* devised a cup to be filled with saline and fitted over the anterior surface of the eyeball to change the error to spherical, but

this cup cannot be worn constantly and is apt to be irritating. The best we can do is to determine the combination of concave sphericals, cylindricals, and stenopaic slits that gives the best vision. Fox's observations show that the size and shape of the stenopaic slit needed varies with the individual eye, and that the choice demands very careful investigation. The correction of the astigmatism is almost equally difficult. Sometimes the best results are obtained by high cylindricals crossed at an oblique angle.

A very important part of the treatment of weakly children with keratoconus is to have them give up all near work, and to live out of doors as much as possible.

When the best vision to be obtained in this manner is much below 20/200 the question of **operation** must be considered. Many operations have been devised to flatten the cone and make the tissue more resistant. The procedure most widely practiced at present is to perform an optical iridectomy, which lowers the tension at the same time, and to cauterize the apex with the galvanic cautery, deeply but not through the entire tissue. The eye should then be bandaged for several weeks.]

In **keratoglobus**, or cornea globosa, all portions of the transparent cornea are bulged forward so as to change its form into that of a hemisphere. This condition is a local phenomenon of the general enlargement of the eyeball met with in hydrophthalmus or buphthalmus. In other words, it is a part of the total sclerectasia that is produced solely by an increase of tension during the early years of childhood, while the sclera is still yielding, and is therefore one of the symptoms of infantile glaucoma.

[Some writers maintain that keratoglobus occurs independently of buphthalmus. A few cases have been reported in which it was met with in eyes that were otherwise healthy and possessed of good vision, and appeared to be a malformation not dependent on disease.]

The nature of **buphthalmus** is not perfectly clear. When congenital it may be due to an occlusion of the natural exits for the intraocular fluid from the eye. The entire eyeball often becomes enormously enlarged, and the sclera so thin that the chorioid shows through, giving it a bluish hue. The enlarged, hemispherical cornea is usually hazy, or opaque like ground glass. The anterior chamber is very deep.

Prognosis

The prognosis of buphthalmus is very bad. Left to itself the

disease goes on to total blindness, and the production of a deep, glaucomatous excavation of the optic papilla, though in rare cases its progress may be arrested at a certain point.

Treatment

A broad iridectomy, or a sclerectomy, should be performed as early as possible. [No form of treatment has proved very successful. Meioties are used by some surgeons, decried as useless by others. Some good results have been reported as obtained by paracentesis, sclerotomy, sclerectomy, and extraction of a lens luxated into the anterior chamber, as well as by iridectomy, but the final enucleation of the enormously distended eyeball is too often the inevitable result of the progress of the disease. A keratoglobus which is a simple malformation, and not a symptom of infantile glaucoma, needs no treatment.]

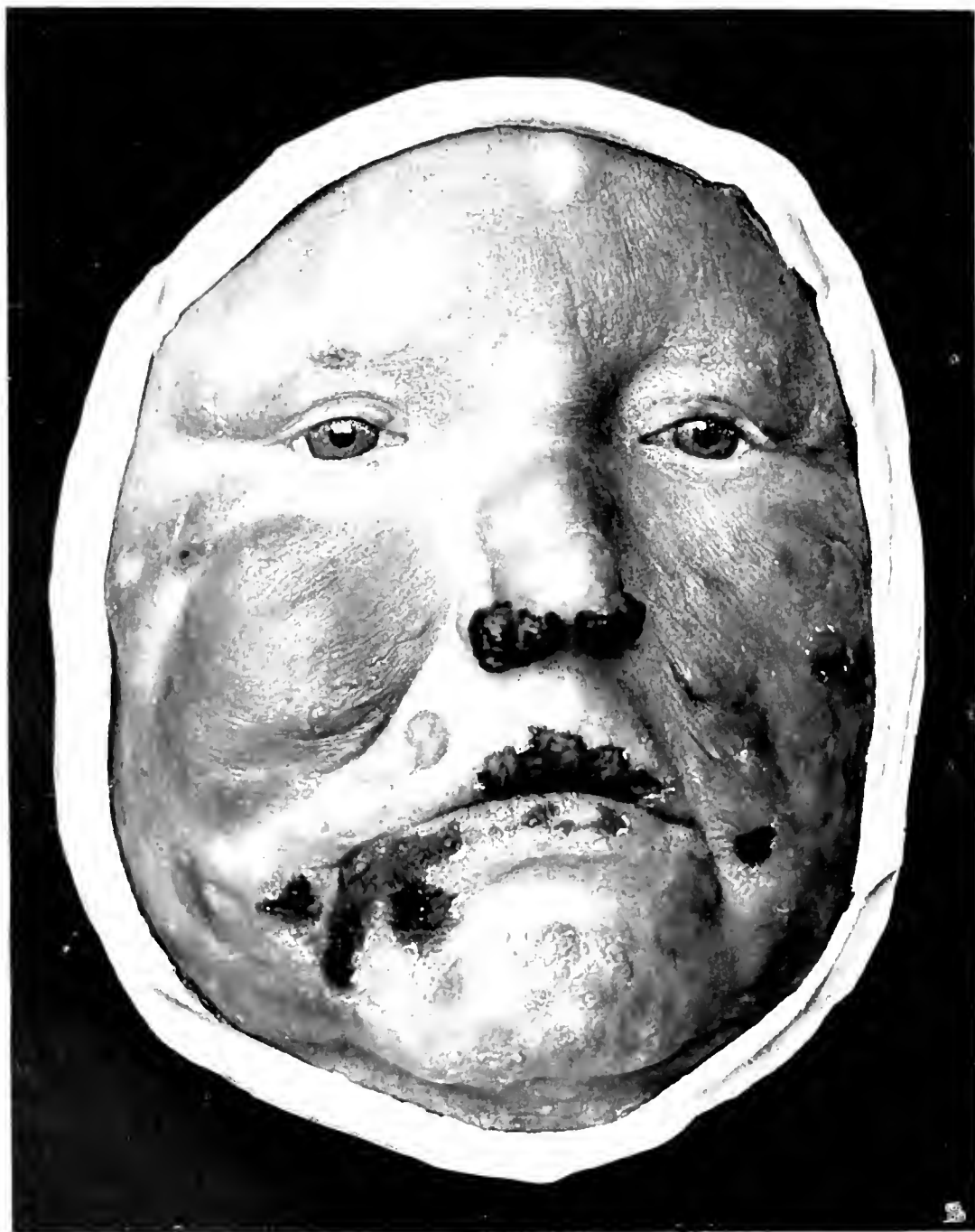


Fig. 60.
Leprosy. Keratitis punctata.



Fig. 61.
Lepra. Epipulbar Leprome.

Leprosy

Plate XLII, FIG. 60; Plate XLIII, FIG. 61

Lyder Borthen emphasized as an important fact that the outbreak of the nodular, or tubercular, form of **leprosy** usually begins in the **eyebrows**. The nodular eruption is also more common than the diffuse infiltration as the initial symptom in this situation. The whitening and falling out of the supercilia is often the first evidence, and may be for years the only sign, of the presence of this disease. The cilia likewise are regularly affected; there is a partial or total atrophy, and the lashes fall out.

The skin of the eyelids frequently becomes involved early, and may present either the infiltrated or the nodular form of leprosy. The infiltration often has an oedematous appearance, and may be continuous with that of the eyebrows, or may be separate from it and appear along the margin of the lid. The nodules are particularly large and are commonly situated along the free margin of the lid. A peculiar feature is their perfectly symmetrical arrangement.

The maculo-anaesthetic form of leprosy occurs quite often in the eyebrows, though not as commonly as the nodular.

Diagnosis

The diagnosis may be determined in any suspicious case by incising an infiltrated place in the skin, squeezing out some of the juice, and examining it for the bacillus of leprosy.

The disease seems to start in the middle portion of the skin. As a rule we can find in the center of the nodule either a lymph vessel, or a blood vessel, very often a capillary, so we know that the bacilli are distributed by both the lymph and the blood. They can frequently be found in the cells of the intima of these vessels, less often in the leucocytes in the lumina. When a capillary or small vessel is the starting point of the nodule it becomes greatly dilated by the engorgement of blood that accompanies the invasion of the bacilli. Infiltration in the vicinity of the vessel appears later, when the bacilli have begun to proliferate outside of the vessel walls, and to excite an

accumulation of wandering leucocytes, together with a great increase of nuclei through the proliferation of the fixed cells of the connective tissue.

The bacilli attack the cells which lie nearest to the vessel and thus carry on the further growth of the nodule. The virus of leprosy is, however, not as rapid in its action as that of tuberculosis, which kills the cells in a comparatively short time, so we may see in a leprous nodule many bacilli in cells that have a normal appearance and have not lost their power to proliferate. One result of this slight tendency on the part of the bacilli to destroy the cells is that leprous proliferations may assume the appearance of tumors.

The *eyeball* is involved very often, indeed almost always. The most common lesion is a punctate, or nodular keratitis, in which little, white, or gray nodules develop beneath the epithelium and cause very little irritation (see Fig. 60). They are composed chiefly of accumulations of leprosy bacilli.

Interstitial keratitis with iritis is not uncommon. [Sometimes multiple, rarely single, nodules appear in the iris and partially fill the anterior chamber.]

Finally the same sort of growths as appear on the lids may occur on the cornea, starting, like other epibulbar tumors, from the limbus (see Fig. 61).

Prognosis and Treatment

[The prognosis is bad. No form of treatment has yet proved effective and reliable. The general policy at present is to isolate the sufferers in colonies.]

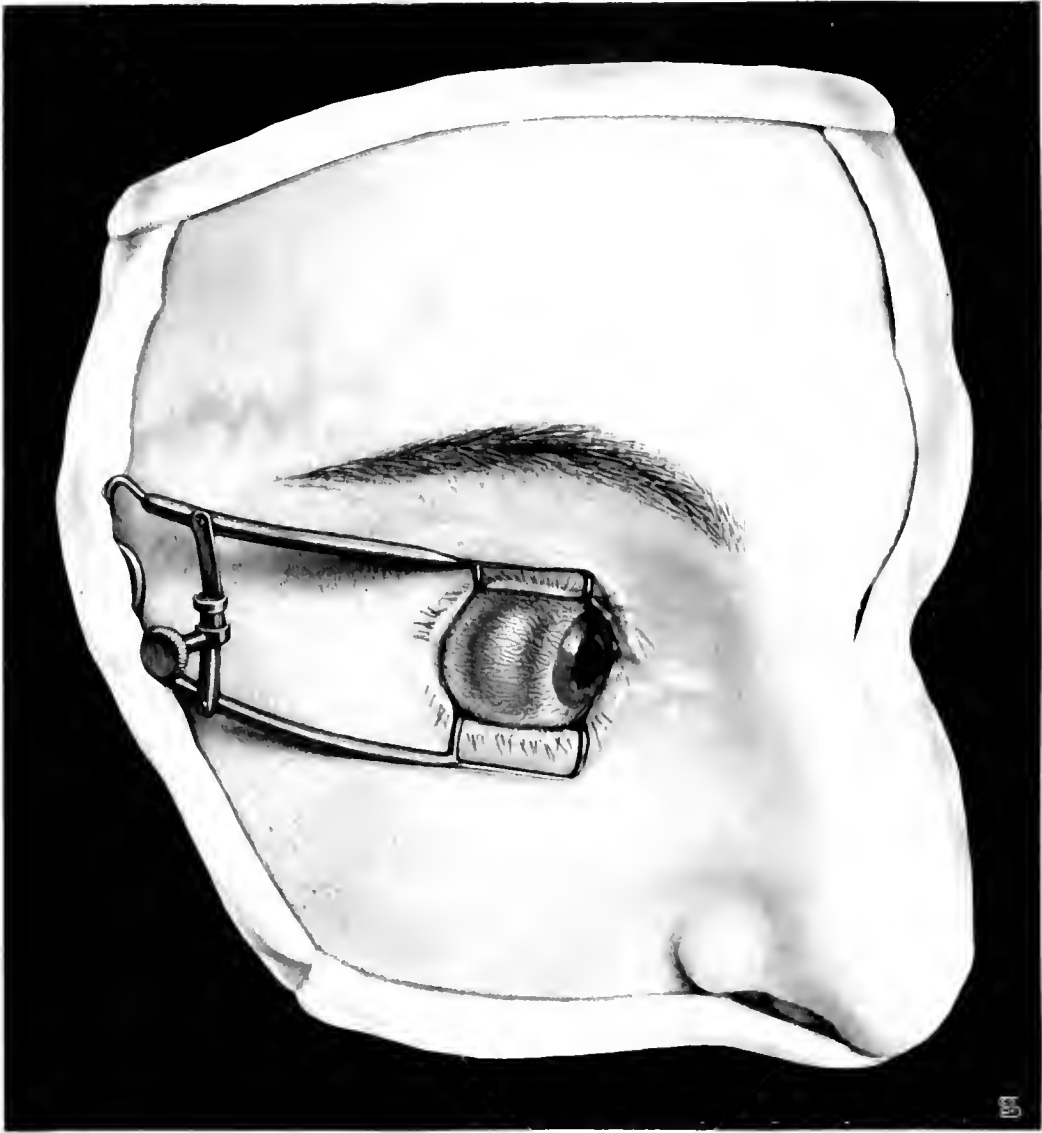


Fig. 62.
Scleritis.

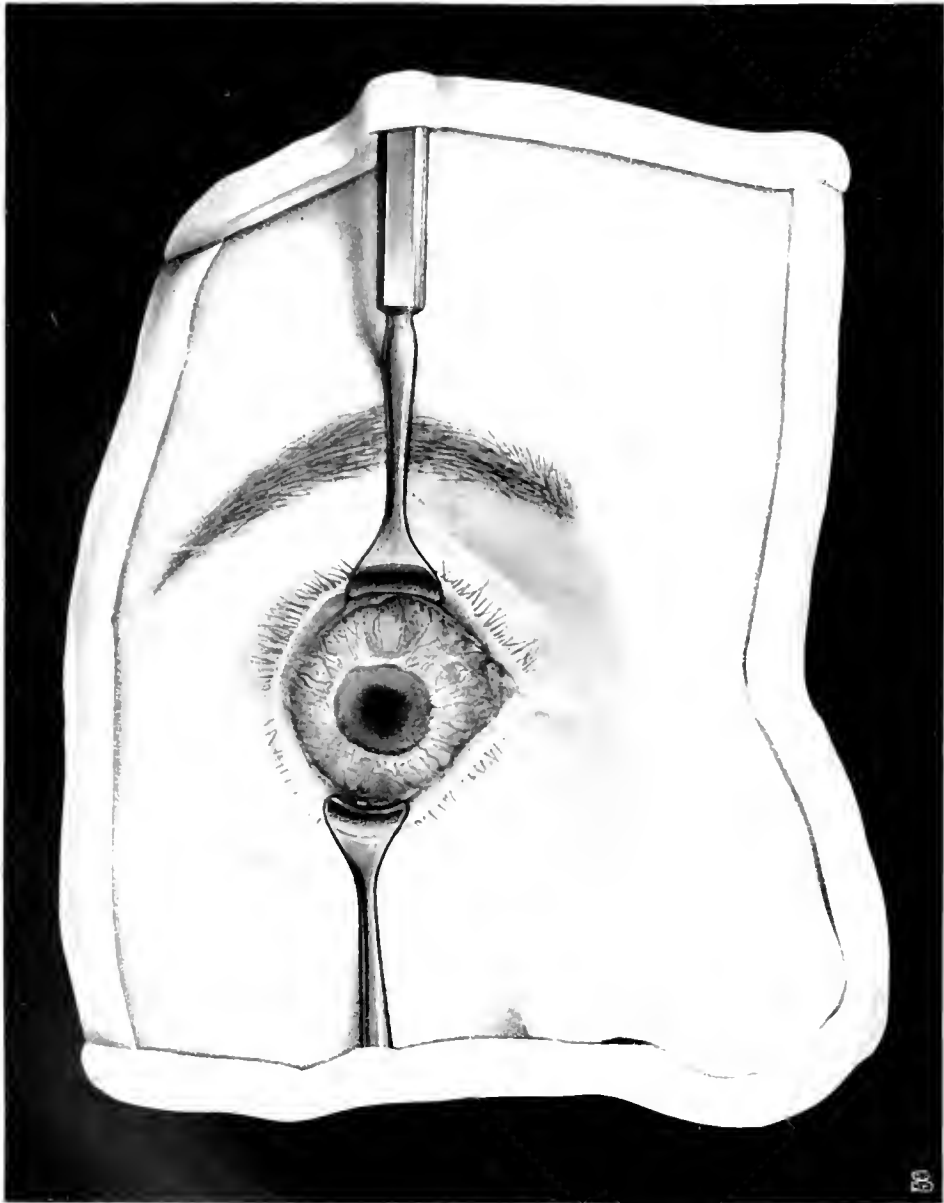


Fig. 63.
Sclerectasia

Scleritis. Sclerectasia.

Plate XLIV, FIG. 62; Plate XLV, FIG. 63

Scleritis is the name applied to a very chronic inflammation of the sclera, met with in persons of middle or old age, and often due to gout, tuberculosis, syphilis, or rheumatism. It is divided into two clinical varieties, the superficial, known as **episcleritis**, in which the superficial layers of the sclera alone are affected, and the deep, or true scleritis, in which the middle and deep layers also are involved.

In the **superficial** form we find a circumscribed focus of disease not far from the cornea, consisting of an exudate that forms a more or less abrupt protuberance, which at first is dark red, later bluish or violet in color, is hard, immovable and very tender to the touch. Dark red, deep, episcleral vessels can be seen running toward and into it, while over it the hyperaemic conjunctiva can be moved. Otherwise the eye may be almost without irritation (see Fig. 62).

[The subjective symptoms vary. Sometimes the patient complains of photophobia, lachrymation, and a more or less violent pain in the eye, while other patients have but slight discomfort.]

The infiltration shows very little tendency to break down or ulcerate. On the contrary it becomes absorbed sooner or later, yet it is evident that more or less of the fibers of the sclera have been destroyed, because, after the inflammation has run its course, a delicate, smoky gray spot is left to mark the place where it existed, or a slate gray spot when the inflammation was more intense. This discoloration is caused by a cicatricial thinning of the sclera which allows the black choroid to show through. This is usually the only injury the eyeball suffers, so the superficial form is much less serious than the deep.

[The mildest form of episcleritis was described by *Fuchs* as **episcleritis periodica fugax**. With the same subjective symptoms a hyperaemia appears in a circumscribed spot on the sclera and in the overlying conjunctiva, but without the characteristic protuberance, lasts for a few days and recurs at intervals.]

Deep scleritis is less common and is not always easy to recognize at first, because we are unable to see how deeply the inflammation has penetrated into the sclera in the absence of a microscopic section. The diagnosis is soon made clear, however, by the never failing involvement of other membranes of the eye, and the onset of an iritis, a chorioiditis, or a sclerosing keratitis. Because of these complications many authors call this form *scleritis complicata*.

We see at first, as in episcleritis, a dark red, or bluish protuberance at a certain place in the sclera, but usually it is not as sharply defined, and it exhibits a marked tendency to reduplication, so that often the entire cornea is finally surrounded by a row of these nodules. In this form also there is no direct breaking down of the tissues, but, after the exudate has been absorbed, the sclera at the site of the inflammation is left cicatricial and greatly thinned, so that the subjacent chorioid gives it a bluish color. In many cases its power of resistance to the intraocular pressure at these points is so much reduced that it yields and permits of the formation of ectasias, or staphylomata.

Such anterior scleral staphylomata are pictured in Fig. 63. They often surround the cornea, when the condition is said to be one of annular staphyloma, but the individual protrusions never become quite confluent, they are always separated by depressions so that they may present an appearance that has been compared, not inappositely, to that of the large intestine.

The eye is held open in Fig. 63 by means of Desmarres' blepharostat, in Fig. 62 by means of v. Graefe's speculum.

Diagnosis

[The diagnosis is not difficult in well developed cases, but in the mild ones of episcleritis the disease may be mistaken for phlyctenular conjunctivitis. The age of the patient is of value; phlyctenular conjunctivitis is usually a disease of childhood, scleritis of middle or old age. In the former the inflamed spot is movable with the conjunctiva over the sclera, in the latter it is not. The top of a phlyctenule soon breaks down and the course of the disease is short; in scleritis the tissue does not break down and the course is long. A drop of adrenalin will cause the redness of a conjunctivitis to disappear, while the deep violet vessels of the episclera are not affected so readily. Finally if the current attack of scleritis is not the first, one or more typically discolored spots may be seen near the margin of the cornea.]

Prognosis

The prognosis in the superficial form is fairly good, except that the duration will be long, [and that recurrences are very common and cannot be guarded against.] In the deep form many eyes are lost through occlusion of the pupil, secondary glaucoma, and other complications.

Treatment

We know of no treatment that is very efficient. The general treatment is important, as it is directed against the constitutional disease, [which is as a rule either gout, rheumatism, syphilis, or tuberculosis. A thorough physical examination should be made in every case to determine which of these is fundamental, and then the treatment indicated should be pushed vigorously.]

Locally the pupil should be kept dilated with atropine [if the iris or cornea show signs of involvement, but the drug should be discontinued whenever the tension becomes increased.] Hot, moist compresses and subconjunctival injections are useful. Massage of the nodules with mercurial ointment is also to be recommended.

[Astringents and caustics must be avoided, but sometimes the application of the actual cautery has proved beneficial. *Darier* states that when the pain is severe it may be relieved frequently by dionin, and that this drug seems to be particularly efficient at the commencement of a gouty episcleritis and in episcleritis periodica fugax. He also recommends the instillation of adrenalin 6 to 8 times a day, and the practice of a gentle, prolonged, rotatory massage of the infiltration with some ointment, preferably mercurial, during the period of anaemia thus produced. Ionization of the nodules with salicylic or lithic ions has also been advocated, as well as the constant electric current, to promote absorption. The treatment of deep scleritis, and of sclerosing keratitis, is the same generally as that of cyclitis.]

Should the intraocular tension become increased an iridectomy is indicated.

Iritis.

Plate XLVI, Figs. 64, 65, 66; Plate XLVII, Figs. 67, 68, 69

Iritis is a common and grave disease of the eye. Every general practitioner should be able to recognize and treat it correctly, for a failure on his part to observe its presence, or to institute the right treatment, may entail an irremediable impairment of vision by the formation within a few days of adhesions at the margin of the pupil, a disablement of the patient, and possibly the loss of the eye.

The **objective symptoms** of iritis are:

1. **A pericorneal or episcleral injection** (see Plate XLVI, Fig. 64). The cornea is seen to be surrounded, close to its margin, by a circle composed of violet red, small, deep vessels that radiate outward like the hairs of a brush. These vessels are the ones that run beneath the conjunctiva, upon the sclera, supply the deep layers of the cornea through their marginal anastomosis, and then pass through the sclera at the corneoscleral margin to the root of the iris; the anterior ciliary vessels. In severe attacks the entire eyeball is reddened and not this zone alone, yet this general redness always increases in depth toward the margin of the cornea, instead of decreasing as it does in cases of simple conjunctival redness (see, for example, Plate XXXVII, Fig. 54).

2. **Changes in the pupil.** The pupil becomes small and slow to react very early in the course of the disease, as the result of a spasm of the sphincter. If nothing is done now to prevent, fibrinous exudations take place on the posterior surface of the iris, and adhesions, called **posterior synechiae**, are formed with the anterior capsule of the lens, first in one place, then in several, and finally about the entire pupillary margin. Sometimes these synechiae cannot be detected until after atropine has been instilled, when the non-adherent parts of the margin of the pupil are seen to dilate, leaving the adherent points jutting like teeth into the pupil (see Plate XLVI, Fig. 65; Plate XLVII, Figs. 67 and 68).

[When the entire margin of the pupil has become bound down to the anterior capsule of the lens, a condition known as seclusion,



Fig. 64.
Iritis. Papulous Syphilitic.

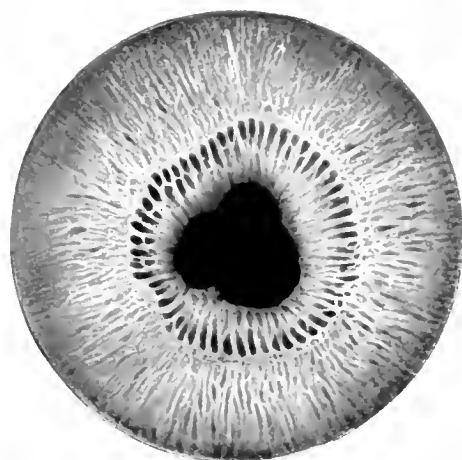


Fig. 65. Synechiae posteriores following Iritis.

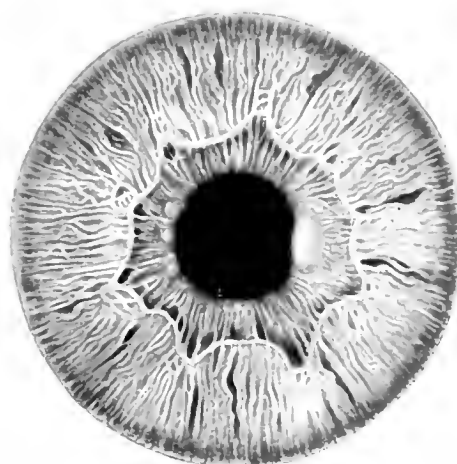


Fig. 66. Condylomata iridis.

or exclusion of the pupil, the passage-way between the posterior and the anterior chambers of the eye is closed, aqueous accumulates in the former, and bulges the iris forward so as to form what is called a **crater pupil**. The displacement of the iris forward thus caused is apt to partially or completely close the filtration angle of the anterior chamber and thus precipitate an attack of glaucoma. In some very bad cases of iritis the entire posterior surface of the iris becomes adherent to the lens, so as to practically obliterate the posterior chamber; this is known as **total posterior synechia**.]

Finally, a deposit of fibrin may fill and close the pupil completely; this is called occlusion of the pupil by an inflammatory membrane (see Plate XLVII, Fig. 69).

3. **Changes in the iris.** The **color** of the iris is often changed so that blue becomes a dirty green, and brown assumes a rusty, reddish tone. [It loses its luster, becomes muddy in appearance, and vessels may be seen running over its surface when a careful examination is made with oblique illumination and a magnifying glass. This change of color and loss of luster are due in part to the hyperaemia, in part to the turbidity of the aqueous, which may be slight, simply imparting to it a yellow hue, or may be considerable. When the inflammation is very severe blood may exude from the iris and may collect at the bottom of the anterior chamber to form a hyphaema.]

In some cases, but not in all, characteristic **nodes** appear on the surface of the iris. Sometimes they are multiple, are situated at the margin of the pupil, and are of an orange red color because they are so vascular; these are considered to be syphilitic papules or condylomata (see Plate XLVI, Fig. 66). In other cases they are pale gray, not very vascular, and distributed along the minor circle of the iris, or are situated in the angle of the anterior chamber; these are tubercles (see Plate XLVII, Fig. 68). **Gummata** usually appear as large, yellow nodes, and are generally single (see Plate XLVII, Fig. 67).

4. The **cornea** frequently presents a delicate steamy opacity.

The **subjective symptoms** of iritis are photophobia, lachrymation, and pain. The last is sometimes severe and radiates from the eye to the temple and brow. [This pain may or may not be very intense, but it is apt to be worse in the early hours of the morning.]

The onset of an iritis may be either insidious or violent. Its gravity constantly increases, and its course may extend over weeks or months, depending greatly on the treatment.

Diagnosis

[The diseases with which iritis is, perhaps, most often confounded are acute catarrhal **conjunctivitis**, and **glaucoma**, yet such a mistake is fraught with great danger, as the treatment of either of these diseases is distinctly contraindicated in iritis. In conjunctivitis the deepest redness is far from the cornea; in iritis it is at the corneal margin. In conjunctivitis the pupil is normal and reacts readily to light; in iritis it is small, sluggish or immobile, and when dilated with a mydriatic is apt to reveal synechiae. In conjunctivitis the iris is of a normal color and appearance; in iritis it is muddy, indistinct, and often of a changed color. In conjunctivitis there is a free mucous or purulent discharge; in uncomplicated iritis there is lachrymation with little if any mucus. In a doubtful or complicated case the presence or absence of iritis can be determined by the instillation of a mydriatic; if the pupil dilates promptly and smoothly there is no iritis, but if it dilates sluggishly, above all, if in dilating it reveals posterior synechiae, the diagnosis is positive.

Glaucoma and **iritis** are so unlike that a differentiation would seem to be unnecessary were it not that they have been mistaken for each other so many times. The only symptoms that may be common to them are a redness of the eyeball, pain, and reduced vision. In glaucoma the eyeball is hard, the cornea insensitive, the anterior chamber shallow, the pupil dilated; in iritis the tension of the eyeball is usually not far from normal, the cornea is sensitive, the anterior chamber is as deep as normal, or perhaps deeper, the pupil is contracted. The age is not a safe guide, but iritis is the more frequent before 45 years of age, glaucoma the more common in later life.

The ciliary body is involved more or less in all cases of iritis, but when this involvement is particularly great we speak of a **cyclitis**, or, better, of an **iridocyclitis**. The characteristic symptoms which distinguish this from an ordinary attack of iritis are an oedema of the upper lid, great pain and tenderness of the eyeball, a marked change in the intraocular tension, which may be either decreased or increased, deposits on Descemet's membrane, and a considerable impairment of vision due to a cloudiness of the vitreous.]

Iritis is always a symptom of some **grave constitutional disease**. There is no such thing as an idiopathic form.

The most common cause is a fresh syphilitic infection that dates back only a few months, but it sometimes occurs in an old case of syphilis.

The next cause in frequency is tuberculosis, when it is to be regarded as due to metastasis from a gland.

It originates not infrequently by metastasis from a gonorrhoea, and may come on after the urethral lesion has been cured.

It appears as a rare symptom in various infectious diseases, but that it can be caused by a cold, no matter how severe, is very doubtful.

Pathogenic organisms may also be carried into an eye through a wound and produce a traumatic iritis.

[Although tuberculosis is a far more common cause of iritis than it was supposed to be not long ago, and although the part played by gonorrhoea in the production of *rheumatic iritis* is probably much greater than it has been thought to be, the majority of authors are not yet ready to abandon the idea that the morbid conditions popularly grouped under the term rheumatism form a very prevalent cause. The opinion which formerly prevailed, that about one-half of all cases of iritis are rheumatic, is probably erroneous. Acute articular rheumatism and gout contribute only small quota, and it may be urged that these are the only cases which can properly be called rheumatic or uric iritis. But the term rheumatism, as popularly used, includes many obscure, indefinite pains, sometimes associated with deposits in the joints, or the formation of concretions in other tissues, which may be looked upon as symptoms of nutritive disturbances situated at any point in the assimilation of food from its digestion in the intestinal tract to its ultimate metabolism in the cells of the organism, and quite a respectable proportion of the cases of iritis occur in connection with these disturbances. The patients are usually, though not always, elderly. Most of them give a history of indefinite rheumatic pains, but it sometimes happens that this is absent and the diagnosis can be determined only by the finding of a considerable excess of urea in the urine.

Rarely a diabetes insipidus may point to assimilative trouble, and the question arises whether such cases do not belong etiologically to the same class. If so, the term rheumatic iritis may be considered a misnomer, although the great majority of the patients present the so-called rheumatic symptoms. These cases are apt to be obstinate until treatment is directed to the nutritive fault, and recurrent attacks are very common.

The diagnosis of tuberculous iritis cannot be established by v. Pirquet's test alone, because this has proved to be positive in almost all adults, irrespective of whether they present any other signs of active tuberculosis or not.]

Prognosis

The prognosis usually is good under proper treatment, for the eye can be saved uninjured in almost all cases, but when the disease is neglected it inflicts permanent injury within a few days, and may even destroy the eye.

Treatment

Atropine is the sheet anchor of treatment in inflammations of the iris. It lessens the quantity of blood, and prevents the formation of posterior synechiae, by contracting the tissue. It is much more difficult to induce mydriasis when the iris is inflamed than when it is in a normal condition, so it is well to remember that the effect of atropine is increased when the solution is warm, and when a little cocaine has been added to it in order to dilate the lymph spaces and so to increase the capacity for absorption. A 1% solution of atropine should be instilled from 6 to 12 times at intervals of a few minutes until the pupil is fully dilated, or until it is evident that it can dilate no more because of the presence of posterior synechiae. The pain generally ceases when mydriasis has been obtained. Cold is borne badly; hot applications on the contrary are very soothing. Heat may be applied in the form of hot, moist compresses, [by bathing the eye at definite intervals for 10 to 15 minutes with hot water, or by means of an electric heater, with which a uniform temperature may be maintained for hours.] When pain persists in spite of the use of atropine and heat, relief may often be secured by the abstraction of blood from the temple. Sweating also frequently has a good effect.

When the pupil is secluded by a circular posterior synechia, an iridectomy must be performed as soon as the inflammation has run its course, in order to guard against a secondary glaucoma.

Above all things the etiology must be determined and the fundamental disease placed under appropriate treatment. [This treatment must be maintained for a long time after the patient has recovered from the iritis and the case has become one of syphilis, or of some other disease, without ocular symptoms. Many patients consult an ophthalmologist instead of their family physicians when their eyes are inflamed, and in such cases it is well for the ophthalmologist to insist that the fundamental disease be investigated and treated from the start by a competent general practitioner, in order that this extremely important general treatment shall **not** be interrupted with the recovery of the eye. As a rule the best results can be obtained only from the co-operation of the ophthalmologist and the general practitioner.]



Fig. 67.
Gumma iridis. Synechiae posteriores

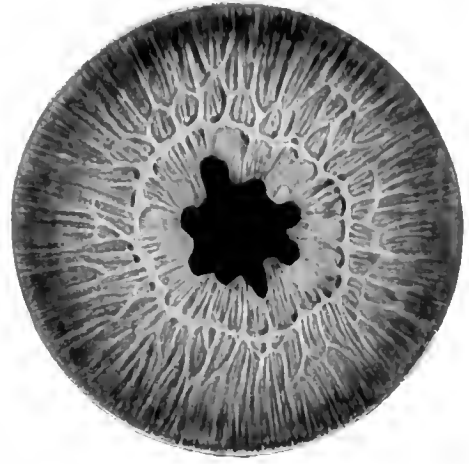


Fig. 68.
Tubercles of the Iris. Synechiae posteriores.

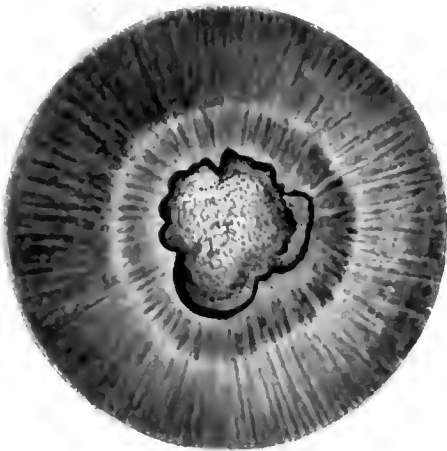


Fig. 69.
Occlusio pupillae
Membrana pupillaris inflammatoria.

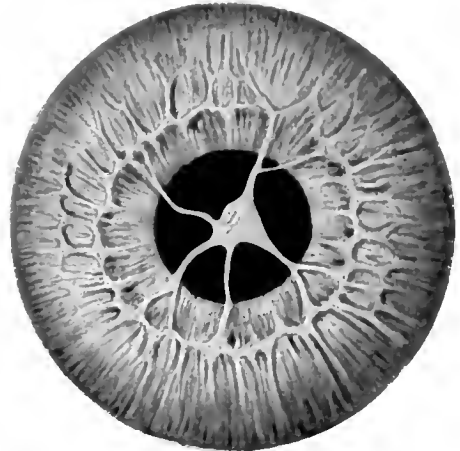


Fig. 70.
Membrana pupillaris perseverans.

Congenital Anomalies of the Iris.

I. CONGENITAL COLOBOMA OF THE IRIS: *Plate XLVIII*,
FIG. 71

II. PERSISTENT PUPILLARY MEMBRANE: *Plate XLVII*,
FIG. 70

I.

A typical congenital **coloboma of the iris** is a cleft, shaped usually like a Gothic arch, directed downward and sometimes a little inward. It is said to be total when the apex of the cleft reaches the ciliary margin, partial when it does not. The corners of its base, where it joins the pupil, are always rounded off, [and never present the sharply defined angles to be seen in artificial colobomata. The fibers of the sphincter are not interrupted.] Sometimes a band of connective tissue passes horizontally across the cleft, forming what is known as a **bridge coloboma**. A strip of tissue without pigment running downward in the iris is called a **pseudocoloboma**.

A typical coloboma of the iris is commonly supposed to be due to an imperfect closure of the fetal cleft, [but the correctness of this theory has been questioned recently by some embryologists, who maintain that the normal fetal iris never has a cleft at any time in its development. Some of the cases reported seem to indicate heredity to be a predisposing cause.

A coloboma of the iris alone seldom injures the vision and is simply a cosmetic defect, but it is apt to be associated with coloboma of the chorioid, or of the optic nerve sheath, or with other malformations in or of the eye, by which the vision may be greatly impaired.]

Numerous atypical colobomata have been described in which the clefts extended in all directions. [A better name for these cases is **partial irideremia**, inasmuch as the commonly accepted view is that a fissure extends downward in the fetal iris, and that a coloboma is due to an imperfect closure of this normal fissure, while a defect in the iris extending in any other direction does not correspond to the position of any normal fetal fissure, but is due to an abnormality

of fetal development. In irideremia the iris is undeveloped to a greater or less degree, so that any portion of it may be wanting, from a small defect in any direction to such complete absence that only rudimentary traces may be found at its junction with the ciliary body. When more or less of the iris is visible the irideremia is partial, when none of the tissue can be seen it is said to be total.

Etiology

The etiology of this condition is obscure; many explanations have been suggested to account for the more or less incomplete development of the iris, but none are perfectly satisfactory. A full discussion of this subject by the editor may be found in the Archives of Ophthalmology, vol. xxvii. no. 6, in which the best suggestion is thought to be a lack, usually hereditary, of sufficient formative material for the development of the eye.]

II.

Persistent pupillary membrane. It is not so very rarely that we see traces of the fetal pupillary membrane in the human eye, which may be differentiated from products of inflammation deposited on the capsule of the lens by the fact that they never start from the margin of the pupil, but always from the anterior surface of the iris, usually from its minor circle.

In the fetal eye the hyaloid artery runs forward through the central canal of the vitreous, or Cloquet's canal, until it reaches the posterior pole of the lens, where it branches and forms a vascular network which covers the entire posterior surface of the lens, and is called the capsular membrane. Branches of these vessels pass to the anterior surface of the lens and there join other vessels that come from the anterior surface of the iris, particularly from the minor arterial circle, and form a membrane in the region of the pupil, called the pupillary membrane. Although traces of this pupillary membrane frequently persist in after life, it is very rarely that any remains of the capsular membrane can be found on the posterior surface of the lens at the same time.

The normal pupillary membrane in the fetus is finely granular and contains oval nuclei irregularly scattered about. Its anterior surface is covered at first by a continuous layer of epithelium, which later always becomes defective. On its posterior surface are the vessels and endothelium. During involution the epithelium disappears first, then the vessels, and finally the membrane itself.

We find in the persistent pupillary membrane a very dense tissue poor in nuclei, with pigment cells scattered about in it, and an endothelial coat, usually incomplete, on both its anterior and posterior surfaces. Its threads are inserted directly into the tissue of the iris, from the structure of which they cannot be distinguished. Its vessels rarely contain any blood.

Glaucoma.

Plate XLVIII, FIG. 72

The essential characteristic of **glaucoma** is an ***increase of the intraocular tension***, which sets in rapidly, as in the acute, inflammatory form, or quite gradually, as in the chronic or simple variety.

Pathologically we commonly find an inflammatory ***occlusion of the filtration angle***, or sinus of the anterior chamber, where the aqueous passes through Fontana's spaces into Schlemm's canal.

[Another characteristic pathological condition, present in all well-marked cases, is an ***excavation of the optic papilla***. This extends to the scleral edge of the nerve head, has an abrupt or seemingly undermined edge, its sides corresponding to the form of the opening through the sclera, and a floor formed by the lamina cribrosa, which is pressed backward. In the early stage the nerve head may be oedematous, but the increased pressure causes the nerve fibers to atrophy, the lamina to yield, and the excavation to be formed.

We also find ***changes in the blood vessels***, the most characteristic of which are to be ascribed to the changes in the papilla. The central vessels are displaced toward its nasal side, and each vessel makes a sudden bend at its edge as it passes from the retina to the side of the excavation, often disappearing from ophthalmoscopic view, as the wall of the scleral opening slants backward and outward, to reappear, apparently disjoined, in the floor of the excavation. The retinal arteries are small, the veins engorged with blood. The only other fairly constant condition is a dilatation of the anterior ciliary veins, which is probably due to a blocking of the vortex veins. The latter are sometimes sclerosed, sometimes not, but their anatomical position is such that they may readily be compressed by the increased intraocular pressure. Schlemm's canal may or may not be partially or wholly obstructed. Arteriosclerosis is often present, as most of the patients are elderly, but it is doubtful if this has usually any connection with the disease, except in the hemorrhagic variety.]

The true **cause** of the increase of tension is unknown. [Many

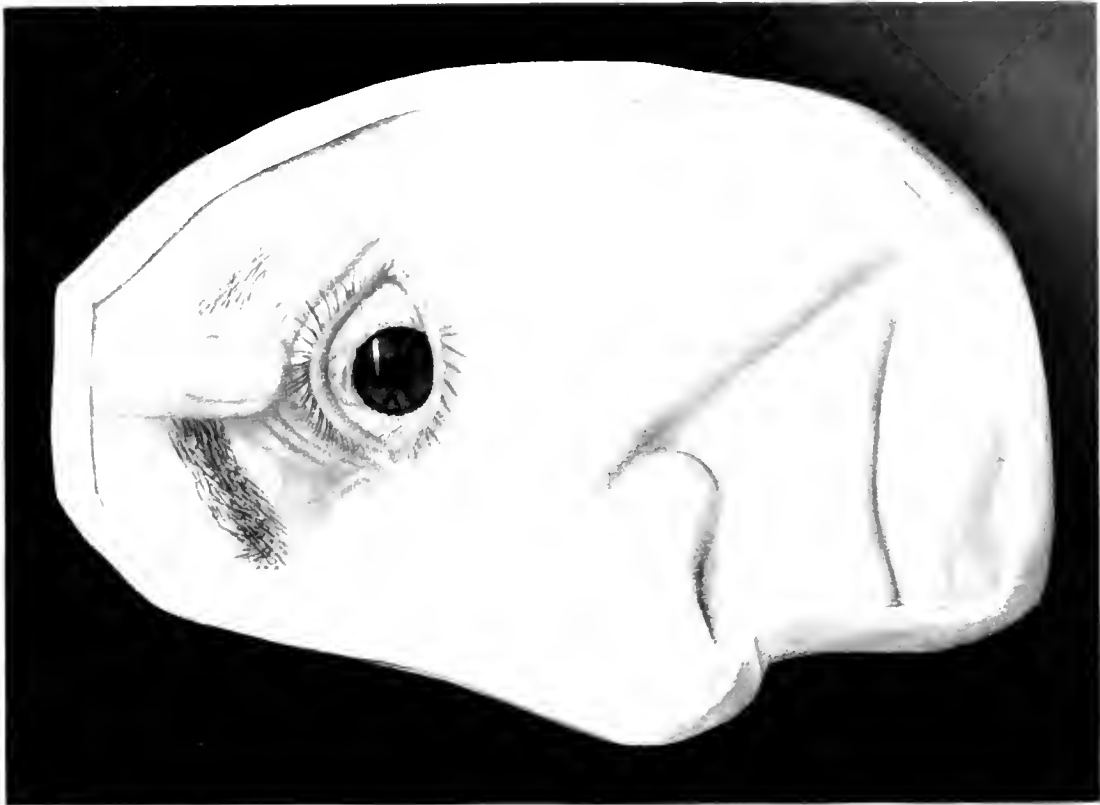


Fig. 72.
Coloboma iridis artificiale
Glaucoma inflammatorum

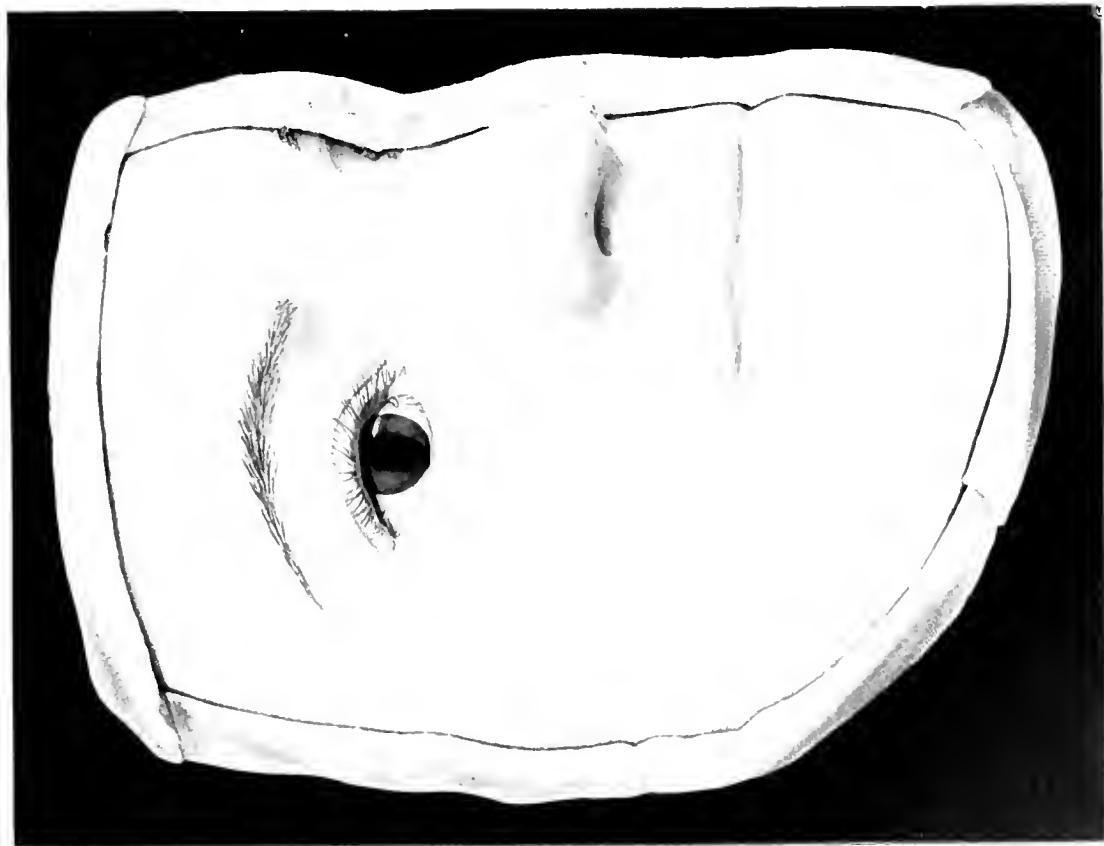


Fig. 71.
Coloboma iridis congenitum

theories have been advanced, and these have been divided by *Roemer* into three classes: first, those in which the increase is ascribed to **retention**; second, those in which it is held to be due to **secretion**; third, those in which it is thought to be caused by a **loss of elasticity in the sclera**. As many of the theories included in these three classes are plausible, and as retention, oversecretion, and loss of scleral elasticity may be present in any combination in a particular case, it is quite possible that there is no one universal cause of glaucoma.

The theory most generally adopted is that advanced by *Priestley Smith*, which belongs to the **first class**. We know that primary glaucoma occurs chiefly in elderly people, and that the lens continues to increase in size till very old age, while the growth of the other parts of the eye ceases much earlier in life, so that the space between the lens and the ciliary processes constantly narrows with advancing age. Hence it is possible that the lens may finally press upon the ciliary processes and force forward the root of the iris until it occludes the filtration angle and prevents the escape of the aqueous. The aqueous thus retained accumulates and increases the hydrostatic pressure in the eye, in other words the intraocular tension. This theory finds support in the fact that glaucoma usually attacks hypermetropic eyes, which are smaller than the emmetropic or myopic, although their lenses are of about the same size.

Other theories of the first class, such as that of a chemical change in the aqueous that excites an inflammation which occludes the outlet passages, and that of a distention of the perichorioidal space, have not been accepted so widely. The weak point in all the theories of this class is that no one can say with certainty that the occlusion of the filtration angle is not secondary rather than primary, and so the result instead of the cause of glaucoma.

The **second class of theories**, that of secretion, refers glaucoma to an increased secretion of fluid in the absence of a compensating excretion. It has been proved experimentally that such a hypersecretion can be excited by stimulation of the sympathetic and trigeminal nerves, as well as by occlusion of the vortex veins. Based on these facts many authors have sought to explain glaucoma as the result of irritative conditions of certain nerves, or as the result of changes in the blood vessels. Some have thought angiosclerosis to be the primary cause. Although much evidence can be adduced in favor of this class of theories, they are not entirely satisfactory by themselves.

According to the **third class of theories** the sclera has lost so much of its elasticity that it can no longer accommodate itself to the fluctuations in the volume of the contents of the eyeball which take place normally. When the sclera is in such a condition it cannot yield as it should when the volume becomes increased, and the tension consequently rises. The advocates of this theory believe inelasticity of the sclera to be the predisposing cause of primary glaucoma, and that the increase of tension and occlusion of the filtration angle are secondary symptoms.

It should also be remembered that glaucoma may be **secondary** to other pathological processes in the eyeball, such as wounds, incarceration of the iris, staphyloma of the cornea or sclera, crater pupil, a wounded or a dislocated lens, and tumors, and that it may follow the instillation of a mydriatic, especially in elderly people.]

An attack of inflammatory glaucoma is commonly preceded by certain **premonitory symptoms**. From time to time the patient notices obscuration, or cloudiness of vision, and at these times sees circles of rainbow colors about any light toward which he may be looking, symptoms which are produced by a slight increase of tension. This is called the prodromal stage.

Sooner or later comes an acute attack. In acute glaucoma the patient suffers from severe pain, which is often unbearable, and has a marked pericorneal injection, a cloudy cornea, a very shallow anterior chamber, a discolored iris, a spontaneously dilated pupil, more or less impairment of vision, and a distinctly hard eyeball.

[**Chronic inflammatory glaucoma** differs from the acute in that the attacks are less violent, and remit rather than pass away, so that the symptoms are to some degree constantly present. The pupil continues widely dilated, the eyeball harder than normal, and the anterior chamber shallow. The iris becomes atrophic. The cornea is sometimes dull and anaesthetic, but occasionally clears so as to permit an ophthalmoscopic examination. At such times the retinal veins are seen to be dilated and tortuous, the arteries to be small and frequently pulsating, and the papilla to exhibit a characteristic excavation. The vision and visual field fall away progressively until total blindness is reached.

Hemorrhagic glaucoma is a form of the disease in which the increase of tension is preceded by intraocular hemorrhages. The blood vessels are seriously affected, and we find inflammatory and degenerative changes in the uveal tract, as well as, perhaps, in the cervical portion of the sympathetic nerve. Arteriosclerosis is com-

monly present. Its etiology seems to vary from that of other forms of glaucoma. To quote from *Risley* "it is but a local expression of the disease of the general vascular tree, and as such anticipates in blindness the fatal result which in many cases speedily follows."]

All of these inflammatory symptoms are absent in **simple glaucoma**. Even the increase of tension cannot be detected with the fingers as a rule, and the patient complains only of failing vision. The diagnosis can be made best and earliest in these cases by means of the perimeter, as the visual field becomes contracted slowly, but constantly, in the form of sectors from the periphery, usually on the nasal side. When this contraction has become considerable the second positive sign may be recognized,—a glaucomatous excavation of the optic papilla which is visible with the ophthalmoscope.

Left to itself every form of glaucoma leads with certainty sooner or later to total, incurable blindness of the affected eye; **glaucoma absolutum**. All of the fibers of the optic nerve become bent and atrophic at the margin of the deep, abrupt excavation, and a total opacity of the lens is almost always added later.

Treatment

The meiotics, physostigmine or eserine, and pilocarpine, are drugs suited for the immediate reduction of tension. They act only as long as the iris remains not atrophic, i.e., as long as its muscular fibers are able to contract. The effect of these drugs is not permanent, but the increase of tension returns as soon as their use is discontinued; hence they are palliative rather than remedial. **Beware of atropine**—it increases the tension.

A broad, peripheral iridectomy should be performed as soon as possible after a positive diagnosis has been made, as generally we may expect to save the remaining vision by this means. Some authors prefer sclerotomy.

[When we consider how obscure is the etiology of glaucoma, and that we are unable to direct our therapeutic efforts at its cause, we are surprised rather that treatment should be as efficient as it is, than at the fact that it is often unsatisfactory. The therapeutic problems to be solved are not quite the same in all forms of glaucoma. In the acute variety the question is how to reduce the tension most quickly, to relieve the extreme pain, and to preserve vision; in the chronic inflammatory it is how to reduce the tension permanently; in the hemorrhagic how to reduce the tension and check the underlying vascular disease; in the simple how to arrest its insidious

course. Conservative treatment with meiotics is hopeless in acute glaucoma, operative treatment too often disastrous in the hemorrhagic and simple varieties. Neither form of treatment can be called curative, for the disease is likely to progress later in spite of either, but we can relieve the pain and save the vision for years in a great many cases.

In an attack of **acute glaucoma** a broad iridectomy, extending as nearly as possible to the extreme periphery of the iris, should be performed at the earliest possible moment. This may effect a permanent cure if the attack is the first from which the patient has suffered. If the attack is not the first the prognosis is not so good, but the progress of the disease may be delayed for many years. This delay may be still further prolonged by the habitual daily instillation of a meiotic.

A carefully performed, broad iridectomy, including the extreme peripheral limits of the iris, may be curative in the prodromal stage of **chronic inflammatory glaucoma**, but in the later stages of this disease the best results are usually to be obtained by a judicious combination of the conservative and operative methods of treatment. An operation should not be lightly performed, subsequent to the prodromal stage, because in a few eyes it will be injurious, and in quite a number of others the atrophy of the optic nerve fibers will advance steadily as though nothing had happened. The patients are then apt to blame the operation for their final loss of vision. It is well first to try to reduce the tension and to maintain it at normal by the instillation of a meiotic, usually eserine sulphate in a $\frac{1}{2}\%$ aqueous solution, two or three times a day. Stronger aqueous solutions are apt to irritate the eye after they have been used for some time, but a 1% solution in oil is borne better. *Darier* recommends the addition of 2% of dionin. The surgeon should test the vision and field at regular intervals, and should keep a record of the tension as determined with the tonometer. If the disease shows no sign of advance this treatment may be kept up indefinitely, but whenever it becomes evident that the disease cannot be held in abeyance in this manner the surgeon must choose and perform the operation which, in his judgment, is best suited for the case. The operation does not do away with the need for the continued use of the meiotic; usually it can be considered only as an adjuvant to this treatment.

In **hemorrhagic glaucoma** the dangers of operative intervention are greatly increased. The general treatment should be about the same as that for arteriosclerosis, quiet, plenty of sleep, protec-

tion against strain of the body or of the eyes, absence of excitement of any kind, and attention to any other disease from which the patient may be suffering. Calcium chloride may be given in doses of 3 to 4 grams per day. Locally eserine, or a 1% solution of pilocarpine, should be used to keep down the tension. Some writers recommend subconjunctival injections of sodium citrate or iodate. When these means fail to control the tension, an operation becomes necessary. The surgeon may choose sclerotomy, sclerectomy, or trephining, but sometimes a paracentesis of the anterior chamber suffices as an aid to the conservative treatment, which must be maintained in all cases. Excision of the superior cervical ganglion has not met with much success in glaucoma in general, but it would seem indicated more in this variety than in the others.

There is a wide diversity of opinion concerning the treatment of **simple glaucoma**, which is far from satisfactory. The prognosis is not good, and, no matter how conscientiously he may conduct the treatment, too often the surgeon feels that, perhaps, the result might have been better had he acted differently. Some writers, like *Ball*, urge the performance of an iridectomy in every case in which vision is equal to counting fingers at one foot, while others claim that this is harmful when the disease is far advanced.

A tabulation of the results obtained by a number of surgeons, compiled by *Roemer*, shows that deterioration followed operation in 66.3%. At the same time the results of conservative treatment are scarcely better. It may be correct to say that very material benefit may be secured by an operation performed in the early stage of the disease, before the fibers of the optic nerve become sharply bent over the edge of an excavation, but no positive method of permanently arresting the course is yet known. The majority, possibly, of surgeons prefer to try meiotics, either arecoline in a $\frac{1}{2}$ to 1% solution, eserine $\frac{1}{2}$ %, or pilocarpine 1%, and to keep a vigilant watch over the visual field, so that if the latter should continue to contract, an operation may be performed without loss of much valuable time. After the operation the meiotic should be continued and the watch over the field maintained. Unfortunately few patients are willing to submit to this necessary control until the disease is far advanced.

Recently the opinion has come to prevail that the essential feature in any operation for glaucoma is the formation of an artificial avenue of escape for the intraocular fluid, i. e. of a filtration cicatrix, although it can scarcely be said to be proved, and although it does not

explain all of the effect produced by an iridectomy. Many operations have been devised to secure this result, of which sclerotomy, sclerectomy, and trephining of the sclera are examples, all of which seem to be superior to the older operation in simple and hemorrhagic glaucoma, as well as in some cases of the chronic inflammatory variety, particularly when the iris is very atrophic. Sufficient time has not yet elapsed to permit a definite judgment as to their final results, but the outlook at present is hopeful.

OPERATIONS

To perform an **iridectomy** for glaucoma a broad keratome is introduced about 1 to 2 mm. behind the upper limbus of the cornea, passed into the anterior chamber in front of the iris until a long incision has been made, and then withdrawn slowly, so as to prevent a sudden escape of aqueous. A pair of iris forceps is then introduced to seize a broad fold of the iris about half way to the pupil, and to draw it from the wound. The iris is then snipped with scissors at the angle of the wound nearest the right hand of the operator, pulled to the left to free the root as much as possible, and then excised with scissors pressing on the sclera. The angles of the wound are then freed from any iris that may be caught in them. Blood is apt to escape into the anterior chamber, and when the amount is considerable a portion of it may be removed by stroking the cornea with a spatula toward the wound, the posterior lip of which is depressed with a Daviel's spoon, but it is not wise to be too insistent on its evacuation lest harm be done. Eserine should be instilled in the *other* eye and a bandage applied. Formerly the unaffected eye was left unbandaged after the operation, because it is so apt to be subject to an acute attack, but the instillation of eserine has proved a more efficient precaution.

Sclerotomy is an incision through the sclera. When performed at the limbus it is called anterior sclerotomy, when back of the ciliary body, posterior sclerotomy. A simple **anterior sclerotomy** is usually made by passing a Graefe knife through the anterior chamber from one side of the cornea to the other at the limbus, and then making an incision upward as though to make a rather small flap for a cataract extraction, allowing the aqueous to escape slowly, and withdrawing the knife before the incision is complete.

Any incision at this place, just back of the limbus, is a sclerotomy, and the latest addition to the number of operations for glaucoma is the **T-shaped sclerotomy** advocated by *van Lint* in 1913 after an

experience of a little more than a year. The conjunctiva is dissected up from all around the upper half of the cornea, exposing the sclera for a distance of at least 1 cm. A suture is inserted on each side of the cornea in such a way that when they are tightened, this conjunctival flap will be drawn down so as to cover the upper fourth of the cornea. The horizontal portion of the **T**-shaped incision is then made with a keratome 2 to 2.5 mm. from the limbus so that the knife enters the eye behind the iris, instead of in front, and generally transfixes it, thus producing an iridodialysis. An iridectomy may now be performed in acute or subacute glaucoma, or a partial, peripheral one in some of the chronic forms, while the iridodialysis suffices in other cases. As the iris is often in contact with the cornea, and as the wound is posterior to it, the iris forceps used should have a tooth on the concave side. One blade of a pair of slender, straight scissors is then introduced into the scleral wound and a vertical cut made from its middle to a point in the cornea 1 to 1.5 mm. from the limbus. At the moment that this is done the lips of the wound separate with force, *van Lint* says, as if some elastic fibers existed at that spot, and a relaxation of the eye is produced. The conjunctival flap is then drawn down so as to cover the entire wound and secured in place by tying the sutures on each side of the cornea. The sutures are removed on the fifth day, after which the conjunctival flap gradually draws back until, at the end of a week, it is in its normal situation. The eye is bandaged for a few days, and a meiotic instilled for some weeks.

Posterior sclerotomy is usually a meridional incision through the sclera into the vitreous between the ciliary body and the equator of the eyeball. Some surgeons add another at right angles, so as to form a **T**-shaped incision. This operation may be performed in acute glaucoma in order to allow the anterior chamber to deepen when it is too shallow to permit of an iridectomy, and has been recommended in hemorrhagic, and in some forms of chronic glaucoma.

Lagrange's operation

In **sclerectomy**, or *Lagrange's* operation, a section is made in the upper quarter of the cornea with a Graefe knife, the incision passing back of the limbus, taking in as large a piece as possible of the sclera, and forming a conjunctival flap. This flap is then laid back over the cornea, and the piece of sclera is excised with scissors, after which the flap is replaced in position. If an iridectomy is done at the same time the operation is called a **sclerecto-iridectomy**.

Elliott's operation

In *Elliott's* operation of **scleral trephining** a large flap of conjunctiva is formed with its base at the cornea, the outer layers of which are carefully undermined for a little distance. This flap is laid back over the cornea, a trephine applied exactly at the limbus and a button of tissue is removed. An iridectomy may be done, or a buttonhole made in the iris. Some surgeons prefer a mechanically driven trephine, others a hand trephine. This operation is the subject of much discussion at present, some writers thinking that it will supersede all others, while other observers point to cases of quiet iritis and of late infection as indicative of its dangers.

Heine's operation

The aim of **cyclodialysis**, devised by *Heine*, is essentially different. Its purpose is to establish communication between the anterior chamber and the suprachoroidal space. After the sclera has been laid bare by an incision through and retraction of the conjunctiva, a cut is made 2 or 3 mm. long through the sclera, parallel to the limbus and 5 or 6 mm. from it, taking care not to wound the chorioid. A spatula is then slowly passed through the wound in the direction of the cornea, pressed close to the inner surface of the sclera, until it appears in the anterior chamber. Lateral movements are then made to detach the iris as much as possible from the filtration angle, the instrument withdrawn, and the conjunctival wound closed with sutures. This operation is very useful as a preliminary step in acute glaucoma when the anterior chamber is too shallow to permit of an iridectomy. In a few cases it may suffice to check the disease, but as a rule it reduces the tension sufficiently to allow the anterior chamber to deepen so that an iridectomy may be performed a few days later with a much better chance of success. It has also been recommended in hemorrhagic and in simple glaucoma, but, as the operation is not ten years old, its indications have not yet been accurately defined.

Excision of the superior cervical ganglion of the sympathetic nerve has not met with much success, although it has produced excellent results in some cases. This is an operation for the general surgeon, and one not to be lightly undertaken, for death has followed in some instances, and annoying complications are not uncommon.]

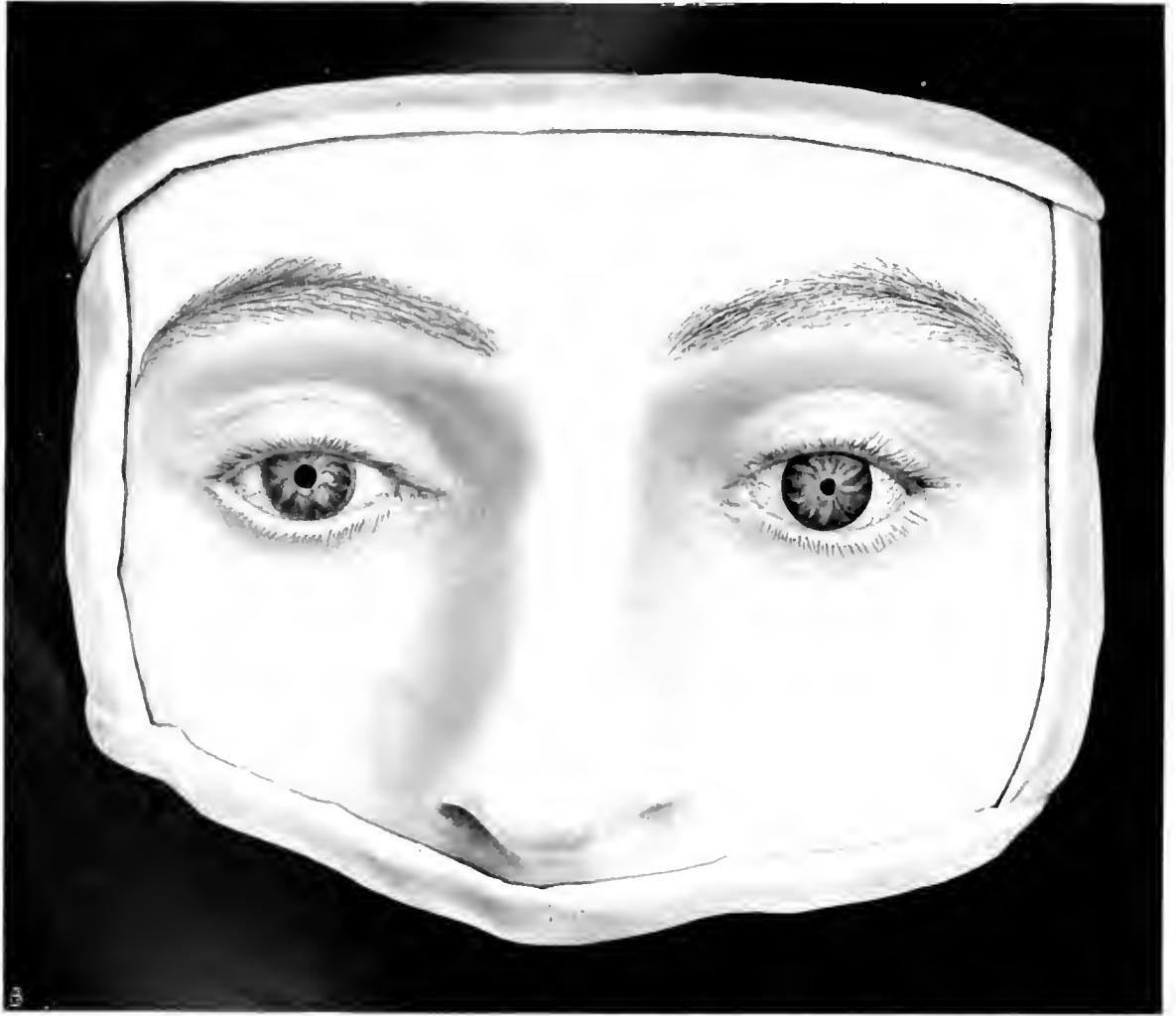


Fig. 73.
Anisocoria. Reaction of pupils in Tabes.

Disturbances in the Motility of the Iris.

Plate XLIX, FIG. 73

Disturbances in the motility of the iris are shown by the behavior of the pupils.

The pupils of the two eyes normally are of equal size. An inequality is called **anisocoria**, and is always indicative of a pathological condition, [except in the rather few cases in which a slight inequality of the pupils is congenital and not associated with any disturbance of their reactions.] One pupil may be in a constant state of abnormal contraction or dilatation, as is often the case in the early stage of cerebral syphilis, or the condition may fluctuate, a pupil suddenly becoming very large or small, an early symptom of progressive paralysis.

The pupils normally contract uniformly to light, no matter into which eye it may be thrown. The contraction of the pupil of the unexposed eye is called the **consensual reaction**, [that of the one into which the light is thrown, the **direct reaction**. The pupils also contract when both eyes are directed toward a near object; this is known as the **reaction to convergence**, or to accommodation.]

The principal tests are those of the reactions of the pupil to light and to convergence. When none of these reactions are present the condition is one of absolute immobility of the pupil. In reflex immobility of the pupil there is no direct or consensual reaction to light, but a prompt reaction to convergence. [In a blind eye there may be no direct reaction to light, although the consensual and convergence reactions are present; or when both eyes are blind all of these reactions are absent; this is the amaurotic immobility of the pupil.

These derangements are met with in a large number of disorders of the general organism, of the nervous system, and of the eye itself, but they are of the greatest diagnostic importance in tabes, cerebral syphilis and general paresis.]

Fig. 73 portrays the eyes of a man 45 years old, who had contracted syphilis 15 years before. Anisocoria is present, the left

pupil is strikingly small (spinal meiosis), and is not perfectly round, a condition strongly indicative of tabes, or of general paresis. The irregular pupil must not be mistaken for one in which the distortion is due to the presence of posterior synechiae of inflammatory origin.

This patient had reflex immobility of the pupils, i. e., both reacted to convergence, but neither to light. His vision and visual field were normal, showing that there was no incipient tabetic atrophy of the optic nerve, but his accommodation was parietic. This latter trouble was the principal reason why he sought the aid of an ophthalmologist. He claimed that he was perfectly well, except for some rheumatism, but the diagnosis of tabes was confirmed by the demonstration of lancinating pains, anaesthetic areas on the legs, absence of the patellar reflex, and the presence of Romberg's phenomenon.



Fig. 74.
Arcus senilis corneae. Cataracta senilis matura.



Fig. 75.
Cataracta morgagniana.



Fig. 76.
Cataracta zonularis

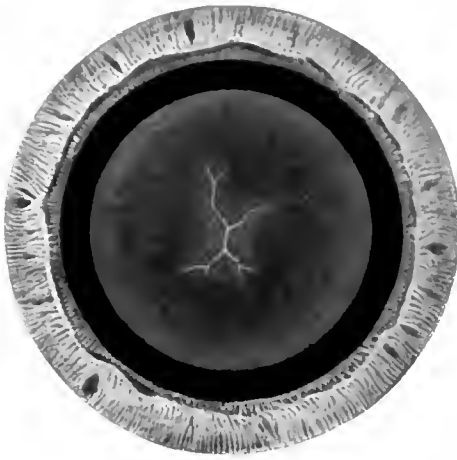


Fig. 77.
Cataracta zonularis with Cataracta stellata



Fig. 78.
Cataracta senilis incipiens.



Fig. 79.
Cataracta capsularis.

Cataract.

Plate L, FIG. 74; *Plate LI*, FIG. 75; *Plate LII*, FIGS. 76, 77, 78, 79

Cataract, derived from the Greek *κατὰ* and *ρήγνυμι* meaning to fall down, is the name applied to the pathological changes in the substance of the lens which manifest themselves as opacities. They depend for the most part on nutritive disturbances, and vary a great deal. Cataracts may therefore be classified in several different ways.

I. We may divide them into the **progressive** and the **stationary**. An example of the former is the senile cataract, which, when once started, is accustomed to progress constantly, though with widely varying degrees of rapidity. An example of the latter is the zonular, or lamellar, cataract, which may not change for decades, or even during a lifetime.

II. We may classify them according to **maturity**. A cataract is ripe when the lens is totally opaque to the capsule and to the periphery. We distinguish the different stages of a progressive cataract as incipient, immature, mature, and hypermature. The final stage is one in which a regressive process has taken place subsequent to maturity, characterized by disintegration, shrinkage, proliferation of the capsular endothelium, calcification, and other changes.

III. We may consider them as **simple** and **complicated**. It would be more accurate to say as primary and secondary, but the term secondary cataract is applied to the more or less thick membrane that is apt to be formed after extraction of the lens, from the remains of the capsule, endothelium, and particles of cortex that have been left behind. A cataract is said to be simple when the eye is healthy, except for the opacity in its lens, and particularly when there is no impairment of the perception of light. A complicated cataract is one in which an opacity of the lens has been secondarily added to some internal disease of the eye that has caused blindness. These two forms present the same external appearance, but they may be differentiated easily by testing the light sense and the projection.

[Light passes through the cataractous lens, simply becoming diffused in its course, so that it is not only perceived by the healthy

retina, but the direction of its source also is instantly recognized. The test of the **projection** is made by casting light from various directions through the pupil; if the light is seen at once and the direction from which it comes is correctly indicated by the patient, the projection is good, but if it is not perceived when it comes from certain directions, the projection is bad and indicates some lesion of the light perceiving apparatus.]

IV. Cataracts may also be classified according to the **age** of the patient into the congenital, soft or juvenile, hard or senile. The congenital cannot always be differentiated from the juvenile, and there is hardly any pathological difference between them. In many cases we have to depend wholly on the history to learn whether the cataract was present at birth, or developed during the early years of life. But there is a great difference between soft and hard cataracts. In youth the lens is of a soft and fairly uniform consistence, and tends to assume a spherical form when removed in its capsule from the eye. In the course of years its central part gradually becomes denser and harder, so that by the time the patient has reached the age of 30 the lens contains a firm, unyielding, and unchangeable nucleus which does not react to contact with aqueous. From that age on the hard nucleus increases in size until it comprises the entire lens. This, when opaque, constitutes the hard cataract, which plays a very important part in operative ophthalmology.

V. Cataracts may also be grouped according to their shapes. In **zonular**, or lamellar cataract, the most common and characteristic form of childhood, an opaque zone appears between the central and the peripheral portions, which remain transparent (see Plate LII, Fig. 77). Sometimes small, densely opaque projections extend from this zone for a short distance into the transparent tissue; these are the so-called "riders" (see Plate LII, Fig. 76).

Central and total cataracts are varieties of the zonular. When the opacity involves only the center of the lens it is called a **central** cataract, not a nuclear, for a nucleus is not present before 30 years of age. When both the center and the periphery are opaque the cataract is **total**. These three types of juvenile cataract shade into each other and present many intermediate forms.

The **total cataract** may also show the following modifications:

Its contents may be fluid and milky, when it is called a *lactate* cataract, or clear, so as to cause the lens to resemble a cyst with limpid contents, a *cystic* cataract. Later not only the layer beneath the capsule, but also the entire contents may calcify, becoming hard

and chalky white, and forming a *calcareous* cataract. Should the fluid contents become absorbed at an early stage, there may remain only a thin membrane composed of capsule and some epithelial proliferations, a *membranous* cataract, or the epithelial cells may calcify beneath the capsule so as to form a thin, stiff, chalky white layer, and create a *papyraceous* cataract. If the capsule should not collapse after the absorption of its contents, it will form a crumpled sac and be known as an *aridosiliquose* cataract.

The fusiform, axial, or ***spindle-shaped cataract*** is a rare congenital form in which the anterior and posterior poles of the lens are joined by a spindle-shaped opacity along its axis.

In ***capsular cataract*** the capsule itself is not opaque, but the opacity is situated beneath it on the anterior surface of the lens. After calcification it is white in color (see Plate LII, Fig. 79). When such an opacity is punctate and situated just at the anterior pole of the lens it is spoken of as an anterior polar cataract; when it is at the posterior pole it is called a posterior polar cataract.

A ***stellate cataract*** is one in which the cement substance of the lens fibers is opaque, so that a figure resembling a star can be seen on the anterior or posterior surface of the lens. Such a stellate figure may be seen at the anterior pole of the zonular cataract shown in Fig. 77, on Plate LII.

Cataracts that appear during middle life often exhibit gray points or lines, and are known accordingly as ***punctate***, or ***striate cataracts***.

VI. Another mode of classification is according to the ***origin***. Thus we have the senile, either cortical or nuclear, which usually begins with radiating opacities that lie between the nucleus and the cortex, start from the periphery, and press forward toward the center until a picture is produced which resembles the spokes of a wheel (see Plate LII, Fig. 78). An opacity of the nucleus, which is of a brown red color (see Plate LII, Figs. 78 and 79), may be clearly distinguished from this gray opacity of the cortex, which later becomes more uniform (see Plate L, Fig. 74).

Black cataract is a variety of the senile and is due to an excessive sclerosis of the nucleus. The pupil looks black, although the lens is opaque.

A ***hypermature senile cataract*** in which the cortex has become fluid, so as to allow the brown nucleus to float about in it, is called a *Morgagnian* cataract. The nucleus sinks into the lower part of the fluid when the patient is standing (see Plate LI, Fig. 75).

Certain **general diseases**, particularly diabetes and nephritis, are sometimes complicated by the development of cataract. Some writers claim that the senile variety should be included in this group. Cataracts may also be produced by poisons. The one caused by ergotin presents characteristics that are very similar to those exhibited by cataracts produced experimentally with naphthalin, sugar, and salt.

When the iris is adherent to a cataractous lens the patient must have suffered at some previous time from iritis with the formation of posterior synechiae. Such a condition is known as an **adherent cataract**.

A **traumatic cataract** is one that has been produced by traumatism. [When it follows an intraocular operation it is sometimes called a **post-operative cataract**.]

Treatment

[Before deciding on the treatment in any given case of cataract we have to take into account whether it is progressive or stationary, hard or soft, simple or complicated. If it is progressive we need to determine its stage as regards maturity; if stationary its variety is of importance. The age of the patient tells us whether it is hard or soft, or composed of a soft cortex with a hard nucleus. Whether it is simple or complicated is ascertained by testing the light sense and the projection. Although this test cannot be made accurately in a very young child, we can draw fairly good conclusions as to the visual power of the retina from his behavior when a light is flashed into the pupil. If the perceptive elements of the retina are in good condition the light attracts the attention of the child and he turns his eyes toward it immediately. *Nothing should ever be done*, so far as the cataract is concerned, *when there is no perception of light*, for its removal cannot restore sight, and a faulty projection is nearly always a contraindication to operation, as both patient and surgeon will almost inevitably be disappointed in the result.

Whenever any part of the pupillary portion of the lens is clear we should first ascertain whether useful vision may be secured by wearing glasses that *correct the refractive error* of the eye, for if this can be done operative intervention is contraindicated.

Fairly good, or useful, vision may coexist with a small anterior or posterior polar, or a spindle-shaped cataract, and then the only thing to be done is to correct the refraction as accurately as possible. The same is true when the central part of a zonular cataract is clear.

Vision may be assisted in the same manner for a while in incipient or immature senile cataracts.

We have at our command no medicamental means to cause a congenital or juvenile cataract to dissipate. They are soft, usually stationary and must either be left alone or operated on, but the method of operative treatment is altogether different from that required by the hard, senile variety. When the opacity so occludes the pupil that useful vision cannot be obtained, and yet the peripheral portion of the lens is clear, atropine should be instilled to dilate the pupil and the vision tested with the eye covered by an opaque disk containing an aperture which corresponds in form and position to an artificial coloboma of the iris, to ascertain whether benefit can be obtained by an optical iridectomy. If the result of this test be good, the most favorable site for the intervention may be chosen by turning the disk so that the aperture will come before different parts of the periphery of the lens.

To perform an **optical iridectomy** an incision is made at the chosen point in the limbus with a narrow keratome, the iris is seized near the margin of the pupil with iris forceps, a fold withdrawn and snipped off with scissors. The angles of the wound are then freed from any entangled iris, atropine is instilled and both eyes bandaged.

The best vision obtainable through an iridectomy is necessarily imperfect, so the decision whether to perform it, or to remove the lens, calls for the exercise of a considerable degree of judgment on the part of the surgeon in many cases. It is possible to obtain better vision by the removal of the lens, but this possibility is not always realized in practice.

When the test with the stenopaic disk above described shows that useful vision is not to be expected after an iridectomy the lens must be removed. This may be done by needling, or by linear extraction.

The operation of **needling** consists of making an incision in the anterior capsule with a knife needle passed through the cornea. As long as a lens is soft its substance may be dissolved and absorbed by permitting the aqueous to enter. Care must be taken that the incision is neither too large nor too small. If it is too large it will allow too much aqueous to pass through, when the lens will swell rapidly, induce pain and an increase of the intraocular tension, and necessitate an immediate evacuation of the lens substance. If it is too small it may close before sufficient aqueous has been admitted to accomplish the desired purpose. Needling frequently has to be

repeated several times before all of the lens substance has been absorbed, and then an aperture may need to be made in the membrane formed from the capsule. This way of removing the lens is slow, but is usually considered to be a safer procedure than the more rapid evacuation by means of a linear extraction.

In membranous cataract the contents of the capsule have been absorbed and a simple needling should suffice to form an aperture when the membrane is not too thick and dense, but often it is necessary to excise a piece near the center of the pupil, and this must always be done when the cataract is papyraceous or aridosiliquose. In lactate and cystic cataracts the contents of the lens are evacuated, and subsequently the membrane is needled if necessary.

The technique of **linear extraction** for the removal of soft lens matter varies somewhat in the hands of different surgeons, but in general is as follows:

An incision is made with a keratome in or near the upper limbus, an iridectomy may or may not be performed, a free incision is made in the anterior capsule, and the lens substance is milked from the eye. After all the visible lens matter has been removed the anterior chamber may be very gently irrigated with warm saline to wash out debris and to render any adherent particles opaque, but it is not essential that every particle should be removed; what is left will become absorbed, unless it be a firm nucleus, which must be sought for and removed whenever the patient is over 25 years of age. It is well to note how differently the eye is affected by soft, juvenile lens matter left within it, and by the soft cortex of immature senile cataracts, which is very apt to excite a serious iritis.

The principal advantages of this operation are that an unabsorbable nucleus can be removed, and that time is saved. Hence it has been used chiefly for the evacuation of wounded lenses, and for the removal in young adults of soft cataracts and of lenses for high myopia, but quite a number of surgeons prefer it to needling, even when they are dealing with infants.

Traumatic cataracts are met with in both young and old persons, but in the latter they are difficult to distinguish from the senile variety. Many cases recover spontaneously, particularly in young people, so it is not best to be in a hurry to operate, unless the lens is badly wounded and swollen. The wisest course to pursue in most cases is to instill atropine, bandage the eyes, put the patient to bed, and await developments. Should the lens swell rapidly and cause an increase of tension, linear extraction should be performed with-

out further delay. Sometimes the opacity clears up completely, so far as can be seen; sometimes it remains confined to the periphery of the lens, and then no intervention is needed. In other cases the opacity may be confined to the pupillary portion of the lens, or it may involve its entire substance. Then the lens substance may become absorbed spontaneously in the course of a few months, but if this does not happen, or if the patient cannot wait so long, operation is necessary. In either case a membrane may subsequently require needling. An optical iridectomy can be of advantage only in the rare cases in which the opacity remains permanently confined to the central part of the lens.

In the treatment of **senile cataract** no very great success has as yet attended attempts to arrest its course, or to cause its retrogression in the incipient or immature stage. None of the advertised collyria, and no form of electrical treatment, has proved competent to cure cataract. Senile cataracts sometimes retrogress spontaneously, but so rarely as to afford no grounds for hope to the individual patient. A possible hope for the future lies in Roemer's treatment of cataractous patients with extracts of mammalian lenses, based on the theory that the ordinary senile cataract is a metabolic disease of the lens caused by a cytotoxin, but this treatment is still in the experimental stage. The instillation of a 2% solution of potassium iodide several times a day for months has been recommended, and has seemed to be of benefit in some cases, and a few surgeons have reported good results from subconjunctival injections of saline, or of a 1% solution of potassium iodide. But in spite of all we can do at present the majority of senile cataracts progress to maturity.

The question how to deal with a progressive immature cataract that greatly impairs the vision of an eye must often be decided by the circumstances of the individual case. Generally it may be said that when only one eye is affected and the sight of the other is good, it is well to await maturity, and that when both eyes of a breadwinner are affected so that his earning capacity is lost or much impaired, one cataract at least should be removed. After the age of 60 the entire lens is hard, whether opaque or not, and many surgeons consider it unnecessary then to await maturity, but even before that age the extraction of an immature cataract does not seem to be so dangerous as it was formerly thought to be. The danger lies in the fact that if much of the cortex is left behind it will excite an iritis which may damage or destroy the eye.

Ripening operations have been devised to render the transparent cortex opaque, so that it may be seen more readily when left behind. The best known is *Foerster's*, iridectomy with massage of the lens through the cornea. *Bettmann* introduces a spatula through a wound in the cornea and rubs the capsule with it, either with or without an iridectomy. The anterior capsule may be lacerated to admit the aqueous, and *Jocqs* has advocated the injection of fluid beneath the capsule. All of these procedures are open to criticism as attended by danger, but very recently *Homer E. Smith* has brought forward what he claims to be a safe and efficient procedure—an extensive crucial incision through the capsule with a sharp, specially constructed knife needle. This, he says, is easy of execution, and not only makes immature cataracts operable by the next day, but also enables them to be delivered whole, leaving no cortex. He further maintains that when this is done prior to extraction a secondary operation on a membrane is scarcely ever necessary. A more extended experience with this operation is needed before it will be possible to say whether these enthusiastic claims are justified or not.

When no ripening operation has been performed a very gentle irrigation of the anterior chamber with warm saline, after the expulsion of the lens, washes out some of the debris and renders the rest opaque so that it can be seen and removed more easily.

All of this trouble is avoided if the lens is removed in its capsule, as done by surgeons who are skilled in Major *Smith's* operation, which seems to be particularly well suited to this class of cases.

When one eye contains a mature cataract and the other is normal, it is well to extract the cataract to enlarge the field of vision, and to prevent the eye from becoming divergent, although binocular vision will not be obtained. The operation will afford no help to the patient whose earning capacity has been reduced by loss of the perception of depth, or of binocular single vision; he should be told that his two eyes will not work together afterward. At the same time the ability to perceive objects on the otherwise blind side is a considerable safeguard against accidents. Another advantage is that if the other eye should later be affected in the same manner, the patient will at no time be blind.

When both eyes contain cataracts of about the same degree of maturity it is best to operate on one, and some days later on the other. It is not usually advisable to perform both operations at the

same time, lest both eyes should suffer in case of an accident, or if infection should take place.

Prior to every operation it should be definitely ascertained that no disease exists in the lachrymal sac, and that the conjunctiva is free from pyogenic organisms. The general preparation of the patient is the same as that commonly practiced in general surgery. Surgeons differ as to the local preparation of the eye, some adopting rigorous and irritating antiseptic precautions, others leaving the eye alone, but as a rule it is unwise to irritate the healthy eye. Instruments are commonly boiled, but boiling dulls the edge of cutting instruments, which should be superlatively sharp, so it is well to sterilize these by placing them in absolute alcohol for two or three hours before and after operation, after which they may be rinsed off with sterile water and dried on a sterile towel. Everything that is to touch the eye must be sterile. The hands of the operator should be as clean as he can make them, but he should not wear rubber gloves, as they interfere with the tactile sense of the fingers, upon which much depends.

The eye is cocainized, the patient placed on an operating table, the skin of the face and lids scrubbed with soap and water, rinsed off with sterile water and dried with a sterile towel. Sterile towels are also laid about the head and face of the patient, and the conjunctival sac is washed out with some bland, sterile solution.

In the **usual operation** a speculum is inserted to keep the lids apart, the conjunctiva is seized close to the lower margin of the cornea with fixation forceps, which are given a half turn so as to hold the eye more firmly, a Graefe knife is introduced in the lateral limbus about 2 mm. above the horizontal meridian of the cornea, passed through the anterior chamber to emerge at exactly the opposite point in the nasal limbus, and an incision is then made upward with smooth strokes of the knife, following the limbus closely, until the knife passes out of the cornea beneath the conjunctiva for a little distance, when its edge is turned forward and made to cut the conjunctiva, thus completing the section and forming a conjunctival flap. The fixation forceps are then removed, the patient told to look downward, and an iridectomy is performed.

The capsule is then incised with a cystitome, or a large piece of it is removed with capsule forceps. Pressure is then made on the lower part of the cornea to cause the upper edge of the lens to rise, emerge from the capsule and engage in the wound, and then gradually upward, following the movement of the lens until it is delivered.

Portions of the cortex that remain may be expelled by stroking the cornea upward with a gentle pressure. These particles may also be removed by irrigation, but this procedure is not without danger, and is not advisable in every case. Adherent bits of cortex may sometimes be removed with a suitable spoon, but this maneuver requires a high degree of dexterity, and is decidedly dangerous in unskillful hands. The angles of the wound are then freed from iris, blood clots and detritus are removed from about the wound, the conjunctival flap is spread neatly in place, the speculum is removed, atropine is instilled, and both eyes are bandaged.

When the iridectomy is omitted the operation is called **simple extraction**.

In the hands of skillful surgeons this operation restores useful, or better, vision in about 95% of the cases, yet it has certain disadvantages due to the fact that most of the capsule is allowed to remain in the eye, where it is apt to retain fragments of lens substance. This capsule with the retained debris, together with a proliferation of its endothelium, is apt to produce a secondary cataract, which necessitates a subsequent intervention in a large proportion of the cases, in order to secure good vision. This secondary operation varies from a simple needling to the removal of a portion with a keratome and De Wecker's scissors, according to the density and toughness of the membrane. Tearing of the membrane with a needle is to be avoided, because the result is apt to be unsatisfactory, and there is danger of exciting inflammation.

Major Henry Smith's operation

Extraction of the lens in its capsule would seem to be the ideal operation, but most attempts to do this have proved disastrous. Within the past few years an operation devised by Major, now Lt. Col., *Henry Smith*, of H. M. Indian Medical Service, has attracted much notice. After the section has been made in the usual way, and an iridectomy, if desired, has been performed, the speculum is removed, an assistant lifts the upper lid out from the eyeball with a large strabismus hook, at the same time pressing up the brow with the ring and little fingers of the hand holding the hook, so as to control any muscular contraction, and depressing the lower lid with the thumb of his other hand, the palm of which lies on the patient's cheek. The patient looks upward, in the position of rest, while the operator presses with a smaller strabismus hook along the lower

portion of the cornea to rupture the zonule and press the lens through the section.

The results of this operation seem to be very good in the hands of the expert, but it should never be attempted without a thorough comprehension of the technique, which is not simple, and an equally skilled assistant, upon whom its success depends to a large degree. The danger of losing vitreous is always imminent.

Atropine should be instilled and both eyes bandaged after all of these operations, as indeed after every operation in which the eyeball is opened, except in the case of glaucoma.

After the eye has recovered from the operation, and the reparative changes that follow have been completed, the refraction should be carefully corrected and glasses prescribed for both distance and near. These glasses will be very strongly convex in all cases in which the patient did not formerly have a considerable degree of myopia. Not less than three months should elapse between the time of the operation and that of the final fitting with glasses, and if a prescription is given earlier it should be made plain that the glasses probably will have to be changed in a short time.]

Dermoids.

Plate LIII, FIGS. 80, 81

Dermoids are congenital, cystic tumors with walls that exhibit the structure of the skin, and occur in places where the skin is not normally present. About the eye they are met with chiefly in two places: I, at the corneoscleral margin; II, beneath the skin near the bony margin of the orbit. Rarely they extend from these places deeply into the orbit and cause more or less damage to the eyeball.

I. **Dermoids of the corneoscleral margin** are dense tumors of a porcelain white, or dull rose color, most often found at the outer margin of the cornea between the insertions of the muscles, where they lie deeply embedded, partly in the cornea, partly in the conjunctiva and sclera, and rise but slightly above the surface of the eyeball. They are always congenital, and are so characteristic that they cannot readily be mistaken for any other kind of tumor.

The structure is seen with the microscope to be like that of a piece of skin; an epidermis composed of several layers, with a connective tissue stroma beneath it, containing sweat glands and hair follicles. The surface is often covered with fine, woolly hair.

The origin of these tumors is attributed by *v. Duyse* to adhesions of the amnion with the surface of the eyeball; by *Remak* to a fetal invagination of the epiblast.

Such dermoids are usually small in childhood, the average size is about that of a lentil, but later in life they may grow larger. They should be removed as early as possible because of the increase in size to be expected, as well as on account of the ugly disfigurement of the eye. The eyeball is intact and well formed in the great majority of cases, but in some rare instances, like the following, its development is interfered with to a greater or less degree by the disturbance occasioned by the presence of the tumor.

Schmidt-Rimpler saw in the eye of a calf a piece of skin covered with hair, that began at the inner margin of the cornea and covered the greater part of its surface. The cornea was rudimentary, the iris was adherent to it, and there was no anterior chamber. The lens protruded through the pupil into the dermoid and was constricted.

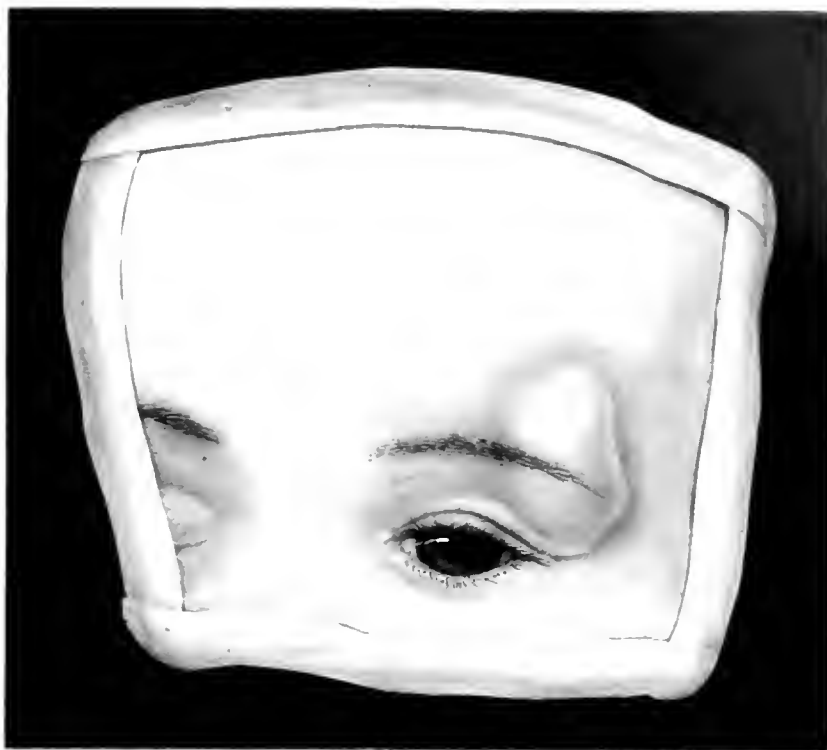


Fig. 80.
Dermoidcyst of the Cornea-scleral margin



Fig. 81.
Dermoidcyst of the orbital rim.

Another interesting case was seen in *v. Graefe's* clinic. A child 8 months old had a congenital tumor of the eye which had nearly doubled in size since birth. It was divided into two portions; one, an ordinary dermoid, covered the entire cornea, with the exception of a narrow edge, and was joined by a short pedicle to the other, which was similar, as large as a cherry, and protruded from the palpebral fissure. The entire tumor was covered by skin with fine hair. The eyeball appeared to contain no lens. Microscopical examination proved that the tumor was a dermoid, and that the skin was provided not only with hair, but also with sweat glands, isolated papillae, and hair follicles. The cornea was absent, its place being taken by connective tissue, to which the iris was adherent. The latter could be recognized only as a thick layer of pigment. There was no anterior chamber.

Bernheimer describes the case of a child 6 months old, who had two tumors, as large as cherries, in his right eye, where they were nearly in contact and kept the lids apart. Later they grew larger and covered the entire cornea. Examination then revealed that the cornea had been so implicated in the dermoid that scarcely a trace was left, and that a total staphyloma had been produced with a proliferation of the iris.

In *Manfredi's* case a well-formed orbit contained a sphere as large as a pea instead of an eye, and the malformation increased somewhat as the child grew. This stump of an eyeball was covered anteriorly with a white, hairy skin, a portion of which was excised and found to present the histological characteristics of dermoid.

Wagenmann examined a tumor that protruded from the orbit of a newborn infant. He found it implanted deeply in the orbit by a pedicle as large as a quill. After division of this pedicle the tumor was removed, and the orbit was found to be lined with mucous membrane, but otherwise exactly like a normal one after enucleation. In all other respects the child was well formed and healthy. Section of the tumor revealed a piece of bone and a rudimentary eyeball. It was encapsuled in skin about 1 mm. thick, covered with fine hair.

II. Dermoids of the skin or subcutaneous tissue of the lids are almost always situated at the outer part of the margin of the bony orbit. They are not very rare, and are usually about as large as peas or plum stones, but may subsequently grow larger. As a rule they are very firmly adherent to the periosteum of the deep surface of the orbital margin, and sometimes they are to be found deep in the orbit. They are congenital cysts with firm walls, and contain a

broth-like substance composed of desquamated, cornified cells and detritus, sometimes with fat and hair.

These tumors are thought to be caused by a deposit of epithelium producing germs at the places affected.

Treatment

[The only treatment is extirpation. When the dermoid lies on the eyeball an incision is made through the conjunctiva and an attempt made to shell out the growth in its capsule, taking care to sacrifice as little of the conjunctiva as possible. A subcutaneous dermoid is removed through an incision in the skin, after it has been dissected free from, or together with the periosteum to which it is adherent. If possible it should be taken out with its capsule unbroken, for then there will be no recurrence, while if a portion of the capsule is left there remains a possibility that it may refill. The operation is not attended with difficulty, except when the tumor is attached to the periosteum far back in the orbit, when the ingenuity of the surgeon may be taxed to perform a complete extirpation and at the same time preserve the integrity of the eyeball.]



Fig. 82. Exophthalmus caused by Tumor retrobulbaris.



Fig. 83.
The Tumor after Exstirpation.

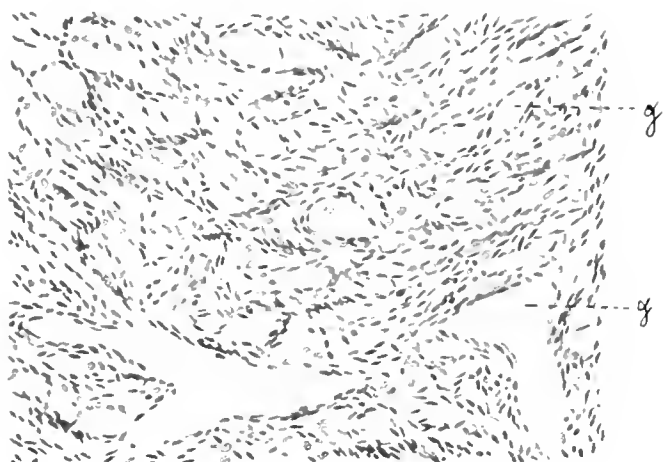


Fig. 84. Microscopic section of the tumor.

Exophthalmus.

Plate LIV, FIGS. 82, 83, 84

By **exophthalmus** is meant a more or less marked projection of the eyeball forward out of the orbit. This condition must never be confused with one in which the entire eyeball is enlarged and therefore protrudes somewhat, although occupying, as a whole, its normal position.

[Exophthalmus is produced mechanically whenever the space within the orbit is diminished by pressure inward of its bony walls, or by an increase in the bulk of its contents, as well as when the muscles that resist the outward pressure of the retrobulbar tissues are parietic. When due to the latter cause it is not apt to be very pronounced, and may be seen not infrequently after tenotomy of the extrinsic muscles.

The eyeball is protruded directly forward only when the driving force is applied exactly behind it; in many cases the displacement is not only forward, but also upward, downward, inward, or outward, and the position of the globe is often a guide to the site of the lesion. For example, when the eye projects forward and outward our attention is directed toward the inner side of the orbit, but it must not be forgotten that a deep-seated pressure acting on the outer part of the posterior wall of the globe may rotate the eye into a similarly divergent position.

When the displacement of the eyeball is the result of inflammation the condition is known as **inflammatory** exophthalmus. The actuating lesion may be a cellulitis, or abscess, in the orbit; an osteitis, or periostitis of its walls; a tenonitis; or a pressing inward of a wall by an inflammation outside the orbit.

Etiology

The cause of a retrobulbar cellulitis, or abscess, may be traumatism, metastasis from a distant focus of inflammation, situated in the parotid gland, the tonsil, or elsewhere, or an extension from a diseased lachrymal sac, but is in most cases an empyema of one or more of the accessory sinuses. One case is on record in which it was

produced by the rupture of an abscess in the zygomatic fossa, caused perhaps by dental caries, through the sphenomaxillary fissure.

An **osteitis** or **periostitis** of the wall of the orbit is likewise commonly caused by inflammation of an accessory sinus, but it may be due to syphilis, and sometimes occurs as a complication in such diseases as measles, scarlet fever, typhoid fever, or tonsillitis.

When the exciting cause is an **inflammation in the accessory sinuses** we find the lids greatly swollen and oedematous, the conjunctiva chemotic, and the eyeball protruding in various directions according to the sinus affected. Frontal sinusitis is the most common, is apt to induce tenderness to pressure on the inner part of the roof of the orbit, which is quite distinct from the sensitiveness of the supraorbital nerve, a bulging of the bone at this place, a mucocele, an osteitis or a periostitis, with the formation of a subperiosteal abscess and a subsequent orbital cellulitis, and thus to displace the eyeball forward, downward and outward. An empyema of the maxillary antrum may cause the eyeball to be forced upward and forward in the same manner, and one of the anterior ethmoidal cells may drive it outward and forward. Exophthalmus is not apt to be caused by a similar inflammation of the posterior ethmoidal cells, or of the sphenoidal sinus, until a cellulitis has been produced, but is more likely to announce its presence by a fairly rapid loss of vision without evident cause.

A **tenonitis** that causes much exophthalmus is usually purulent. It is commonly due to an infected wound, but sometimes occurs as a complication in infectious diseases. A diagnostic feature of tenonitis is that the eye is fixed in position by the pain which attends every attempt to move it.

A simple non-inflammatory exophthalmus may be produced by **emphysema** of, or **hemorrhage** into the orbit as the result of traumatism, is met with congenitally, appears as a symptom in certain general diseases and cases of poisoning, but is most often due to a **tumor**, or other chronic enlargement behind the eyeball in the orbit. It may be more or less stationary, may slowly increase, may be pulsating, or may be intermittent.

Intermittent exophthalmus is due to a vascular abnormality in the orbit, such that when the head is held in certain positions the eyeball is caused to protrude by a venous engorgement, but returns to its place when there is no impediment to the venous circulation. This condition does not seem to be indicative of any serious trouble, and is not amenable to treatment.]

Pulsating exophthalmus is usually due to an arteriovenous aneurysm, caused by a laceration of the carotid artery in the cavernous sinus. The pulsation may be distinctly felt when a hand is laid upon the eyeball, or sometimes it may be seen with the naked eye.

[Exophthalmus is one of the symptoms that compose the syndrome known as **exophthalmic goiter**, Basedow's disease, or Graves' disease. It occurs sometimes in **acromegaly**, perhaps as a result of a proliferation of the orbital fat, perhaps because of a circulatory disturbance dependent on compression of the cavernous sinus. Very rarely it is met with in hydrocephalus, and in tuberculosis when the retrobulbar tissue has been attacked by the disease. In leucocythaemia and pseudoleucocythaemia benign retrobulbar tumors, known as **lymphomata**, sometimes develop and produce exophthalmus. These are the only retrobulbar tumors, except the syphilitic, that have been known to retrogress and disappear under medicinal treatment.

Cases have been observed in which exophthalmus developed after the prolonged use of thyroïdin, and a constituent of some hair dyes, paraphenylendiamin, is known to produce exophthalmus in dogs.

A stationary exophthalmus may be caused by a congenital malformation of the skull in which the orbit is shortened by an abnormal position of the greater wing of the sphenoid.

A slowly increasing, non-inflammatory exophthalmus is generally caused by a retrobulbar tumor, which may be either benign or malignant, and of almost any kind. In some cases a fairly positive diagnosis can be made as to its nature and location, but in many this cannot be done without an exploratory operation.

Treatment

The treatment of an **inflammatory exophthalmus** should always be very energetic, as not only the integrity of the eye, but life itself is threatened. When the cause is not clear, and particularly when there are symptoms present that suggest syphilis, Wassermann's test should be made; if this is positive and corroborative of other symptoms, antisyphilitic treatment should be tried, but if the response is not quick, other measures should be adopted. In cellulitis or periorbitis a free incision should be made so as to allow an unimpeded escape to the products of inflammation, and a thorough investigation then made to ascertain the site of the primary trouble. When there is any reason to suspect traumatism, especially in a young

child, a careful search should be made for a possible foreign body, as fatal results have followed in a number of cases in which this rule was not observed. When the abscess is due to metastasis, simple evacuation and drainage is the only local treatment. When it comes from the lachrymal sac the latter demands immediate attention. In most cases the trouble will be located in one or more of the accessory sinuses, and then the best course is to refer the patient to a rhinologist, for it is not always easy, even for the expert, to decide whether endonasal treatment or a radical operation will be best in the individual case. Many patients can be cured by the former, many require the latter, but in all the treatment must be determined by the sinus or sinuses affected, the intranasal conditions, and other attendant circumstances.

When a simple, *non-inflammatory exophthalmus* steadily increases, the possibility that it may be caused by a gumma must be borne in mind, and a Wassermann test should be made in all cases in which the nature of the lesion is not evident. But a positive Wassermann is not a proof of the syphilitic nature of the particular lesion; any benign or malignant growth may appear in a syphilitic patient. Hence, when a positive test together with other evidence justifies the suspicion that the lesion is syphilitic the appropriate treatment should be given energetically, and a close watch kept on the result. Should the exophthalmus begin to subside, the treatment should be maintained, otherwise other measures should be taken before too much time has been lost.

When an exophthalmus is caused by a growth that is manifestly malignant the entire contents of the orbit should be extirpated as soon as possible. The only exception to this rule is when the disease has evidently involved the adjacent structures to such an extent as to render intervention hopeless either to save life, or to relieve pain. Some hope for the future in the treatment of cancer is given by the researches being conducted at present with regard to the effects of radium, but thus far the best use of radium and x-rays is to irradiate the tissues after operation to ferret out bits of cancer that have been left behind, and so to try to prevent recurrence.

When the tumor is plainly benign and so situated that it may readily be approached, it may sometimes be removed through an incision in the conjunctiva. But in the majority of cases approach is difficult, and the nature of the growth is hard to determine; then it is well to gain access to the orbit by means of Kroenlein's temporary resection of its outer wall. Benign tumors may be removed

through the aperture thus created with a minimum of injury to the eyeball, or the contents of the orbit may be exenterated if the case prove to be one of malignant disease. Even though the growth be such that its removal impairs or destroys the sight of the eye, the preservation of the eyeball is of considerable importance to the development of the side of the face and the appearance of the patient, especially in the young. This is illustrated by the following case, which was operated on by the editor:

A boy, 6 years old, had a moderate degree of exophthalmus forward, upward and inward, caused by a rather firm, smooth, freely movable tumor that could be felt by deep palpation in the lower, outer part of the orbit. Kroenlein's operation was performed and the optic nerve found to be encapsuled in a sac of fluid. This was aspirated and its contents found to be of exactly the same composition as the cerebrospinal fluid. Twelve years later, the eye, though blind, was of normal appearance, the face was symmetrically developed, and the scar was not disfiguring, or particularly prominent.

Kroenlein's operation

Kroenlein's operation is thus performed: A curved incision is made through the skin, fascia, and muscle tissue so as to lay freely bare the outer margin of the orbit. The periosteum is divided along this margin and elevated from the orbital surface of the bone as far as the outer angle of the sphenomaxillary fissure. The orbital plate of the malar bone is cut with a thin chisel through the upper edge of the zygomatic process horizontally inward to the sphenomaxillary fissure, and a second incision is chiselled from this fissure obliquely upward and outward through the external angular process of the frontal bone. The bony flap thus formed is rotated outward with a strong pair of forceps, the periosteum is divided vertically, and access is gained to the interior of the orbit. After the tumor has been removed the edges of periosteum are joined, the bony flap replaced in position, and the skin wound sutured.]

Fig. 82 is the picture of a woman 60 years old whose right eye had gradually become dislocated forward, downward and outward in the course of two years, with no signs of inflammation and no pain. Inflammatory exophthalmus was excluded, for this usually comes on suddenly or rapidly, and is produced by inflammatory exudates, or masses of pus, in the structures behind the eye, very often coming from the accessory sinuses. In such cases the lids are red, swollen and oedematous, and the conjunctiva is chemotic, but nothing of the

sort is to be seen here, aside from a couple of dilated vessels in the bulbar conjunctiva.

It was not a pulsating exophthalmus, and exophthalmic goiter was easily excluded. Evidently the exophthalmus was due to the presence of a retrobulbar tumor, which was probably benign as it had been of very slow growth.

A tumor of the optic nerve is not uncommon, but could be excluded in this case by two circumstances: a tumor of the optic nerve is apt to force the eyeball directly forward, and to destroy the sight of the eye at an early stage through compression of the optic nerve fibers, but in this case the exophthalmus was not directly forward and the vision was fairly good. The optic nerve is curved like an S in its course through the orbit from the optic foramen to the eyeball, and so, in cases of exophthalmus, it can yield a good deal without injury by becoming straightened.

The position of the eyeball excluded any of the comparatively common tumors of the lachrymal gland, for such tumors displace the globe downward and to the nasal side.

The movements of the eye were fairly good, hence the tumor was not adherent to the ball, and probably did not involve to any great extent the immediately adjacent tissues, or the muscles. The nose and all of the accessory sinuses were unaffected, hence this was a primary tumor of the orbit and had not penetrated elsewhere. By pressure with the finger on the upper part of the upper lid between the bone and the eyeball two hard nodules could be felt, and cysts, entozoa, both echinococci and cysticerci, and lipomata were eliminated, as these are soft. In this manner the diagnosis was made by exclusion of a hard, encapsulated fibroma, while its size and location were accurately determined.

The slow but steady growth of the tumor proved its extirpation to be necessary, for as soon as an exophthalmus becomes so great that the lids can no longer cover the eyeball sufficiently, the condition becomes one of great agony.

The operations to be considered when a tumor is present are exenteration of the orbit, with sacrifice of the eyeball, removal through the conjunctiva with preservation of the eyeball, and extirpation after Kroenlein's operation. The first was contraindicated in this case because the tumor was evidently benign and encapsulated, the last was impracticable because the tumor was situated on the nasal side of the orbit, and the following method was adopted.

An incision was made through the bulbar conjunctiva on the inner

surface of the eyeball, the tendon of the internal rectus was picked up, secured with a suture and then divided, a blunt retractor was introduced to draw the eyeball downward and outward, so as to give room for the passage of the tumor. The tumor was hard, about as large as a small hen's-egg, and its only attachment was deep in the orbit by a slender pedicle (see Fig. 83). It was easily removed. The eyeball was then replaced in position, and the cut ends of the tendon were sutured together.

Microscopic examination showed the tumor to be composed of a fairly cellular, hard connective tissue (see Fig. 84). The tumor did not return and the eyeball has remained in good condition.

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